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(54) Title: METHODS FOR DIAGNOSING AND TREATING SCHIZOPHRENIA

(57) Abstract: The genes encoding SCYA2, GADD45B, S100A8, CDKN1A, IL1RL1, TGM2, MAFF, SERPINA3, GRO1, CD14, KIAA1075, CHI3L1, SERPINH1, MT1X, KIAA0620, TIMP1, NUMA1, DDIT3 and TOB2, are upregulated in the anterior cingulate of schizophrenic patients compared to normal patients and as such are useful drug targets for schizophrenia. Methods of screening, diagnosing and treating schizophrenia based on these genes are provided. Transgenic nonhuman animals having increased copy number or increased expression levels of these genes are also provided. The transgenic nonhuman animals are used in methods for screening for potential therapeutic agents.

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METHODS FOR DIAGNOSING AND TREATING SCHIZOPHRENIA

BACKGROUND

1. Field of the Invention

The present disclosure relates to genes correlated to schizophrenia and methods of using genes for diagnosis and treatment of schizophrenia.

2. Description of Related Art

Schizophrenia is a severe psychiatric disorder usually characterized by withdrawal from reality, illogical patterns of thinking, delusions and hallucinations, and accompanied in varying degrees by other emotional, behavioral, or intellectual disturbances. See Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, American Psychiatric Association, 273-315 (1994) (DSM-IV™). However, as stated therein, no single symptom is pathognomonic of schizophrenia; the diagnosis involves recognition of a constellation of signs and symptoms associated with impaired occupational or social functioning. Id. Some detectable physiological changes have been reported, e.g., neuropathological and imaging studies depicting anatomical alterations associated with the disease. Arnold et al., Acta Neuropathol. (Berl) 92, 217- 231 (1996); Harrison, Brain 122, 593-624 (1999). Certain cellular aberrations have been observed and biochemical and RNA analyses have demonstrated alterations in some neurotransmitter pathways and presynaptic components. Id.; Benes, Brain Res. Rev. 31, 251-269 (2000).

At beginning stages and even at more advanced stages, schizophrenia can involve subtle behavioral changes and subtle and/or undetectable changes at the cellular and/or molecular levels in nervous system structure and function. This lack of detectable neurological defect distinguishes schizophrenia from other well-defined neurological disorders in which anatomical or biochemical pathologies are clearly manifest. Thus, there is a need for non-subjective modalities for screening and diagnosis of schizophrenia. Moreover, identification of the causative defects and the

neuropathologies of schizophrenia are needed in order to enable clinicians to evaluate and prescribe appropriate courses of treatment to cure or ameliorate the symptoms of schizophrenia at early stages or when symptoms are obscured. Indeed, there are few effective therapies for the disease and its molecular basis is still not well understood.

Methods have been designed to survey alterations in mRNA expression in order to search for genes disregulated in various diseases and disorders. In organisms for which the complete genome is known, it is possible to analyze the transcripts of all genes within the cell. With other organisms, such as human, for which there is an increasing knowledge of the genome, it is possible to simultaneously monitor large numbers of genes within a cell. DNA microarray analysis is a technique that permits the quantitative measurement of the transcriptional expression of several thousand genes simultaneously. This technique permits one to generate profiles of gene expression patterns in both patients suffering from schizophrenia and control individuals. Accordingly, determination of abnormal levels of gene expression provides a signpost for therapeutic intervention.

Techniques for modifying RNA levels and activities involve ribozymes, antisense species, and RNA aptamers and small molecule promoter modulators. Ribozymes are RNAs capable of catalyzing RNA cleavage reactions, and some can be designed to specifically cleave a particular target mRNA. Ribozyme methods include exposing a cell to, inducing expression in a cell, etc. of such RNA ribozyme molecules. Activity of a target RNA (preferably mRNA) species, specifically its rate of translation, can be inhibited by the application of antisense nucleic acids. "Antisense" nucleic acids are nucleic acids capable of hybridizing to a sequence specific portion of the target RNA, e.g., its translation initiation region by virtue of some sequence that is complementary to a coding and/or non-coding region. The antisense nucleic acid can be oligonucleotides that are double-stranded or single-stranded, RNA or DNA or a modification or derivative thereof, which can be produced intracellularly by transcription of exogenous, introduced sequences in controllable quantities sufficient to perturb translation of the target RNA.

The above described techniques are emerging as an effective means for reducing the expression of specific gene products and may therefore prove to be uniquely useful in a number of therapeutic, diagnostic and research applications for the modulation of genes that are disregulated in schizophrenic patients.

We have previously discovered that three genes (decidual protein induced by progesterone (DEPP), adrenomedullin and cold shock domain protein A (cdsA)) are upregulated in schizophrenia. We have now surprisingly discovered that mRNA for nineteen other genes (disclosed in Table 1 herein) are similarly upregulated in samples from schizophrenic individuals. Thus, these genes can be used as novel drug targets for schizophrenia.

SUMMARY

In one aspect, a method for screening for schizophrenia in a population is provided which comprises determining, in members of the population, the magnitude of expression of a gene selected from the group consisting of those disclosed in Table 1 in a sample and comparing the magnitude of expression to a baseline magnitude of expression of the gene, wherein increased gene expression indicates the presence of schizophrenia. The sample may be taken from the brain, spinal cord, lymphatic fluid, blood, urine or feces.

In another aspect, a method for diagnosing schizophrenia in a host is provided which comprises determining the magnitude of expression of a gene selected from the group consisting of those disclosed in Table 1 in a sample and comparing the magnitude of expression to a baseline magnitude of expression of the gene, wherein increased gene expression indicates the presence of schizophrenia.

In another aspect, a method for treating schizophrenia in a host is provided which comprises lowering expression of a gene selected from the group consisting of those disclosed in Table 1 by administering to the host an expression lowering amount of antisense oligonucleotide.

In another aspect, a method for treating schizophrenia in a host is provided which comprises lowering expression of a gene selected from the group consisting of the genes disclosed in Table 1 by administering to the host an expression lowering amount of a ribozyme which cleaves RNA associated with expression of the gene.

In another aspect, a method for treating schizophrenia in a host is provided which comprises lowering expression of a gene selected from the group consisting of those disclosed in Table 1 by administering one or more nucleic acid molecules designed to promote triple helix formation with said gene.

In another aspect, a method for treating schizophrenia is provided which comprises reducing the amount of a gene disclosed in Table 1 in a patient by administering an effective amount of an antibody against the protein or proteins selected.

In another aspect, a method for treating schizophrenia is provided which comprises reducing the amount of a gene disclosed in Table 1 in a patient by administering an effective amount of a RNAi against the gene or genes selected.

In another aspect, a method of screening for compounds which are useful in the treatment of schizophrenia is provided which comprises operatively linking a reporter gene which expresses a detectable protein to a regulatory sequence for a gene selected from the group consisting of those disclosed in Table 1 to produce a reporter construct, transfecting a cell with the reporter construct, exposing the transfected cell to a test compound, and comparing the level of expression of the reporter gene after exposure to the test compound to the level of expression before exposure to the test compound, wherein a lower level of expression after exposure is indicative of a compound useful for the treatment of schizophrenia.

In another aspect, a transgenic nonhuman animal is provided whose genome stably comprises an increased copy number of a gene selected from the group

consisting of those disclosed in Table 1 wherein the gene is expressed at higher than baseline levels and the animal exhibits abnormal behavior.

In another aspect, a transgenic animal is provided whose genome stably conmprises a gene selected from the group consisting of those disclosed in Table 1 wherein expression of the gene is enhanced by at least one alteration in regulatory sequences of the gene such that the gene is expressed at higher than baseline levels and the animal exhibits abnormal behavior.

In another aspect, a transgenic nonhuman knockout animal is provided whose genome stably comprises a homozygous disruption in one or more genes selected from the group consisting of those disclosed in Table 1 wherein said homozygous disruption prevents the expression of the gene, and wherein said homozygous disruption results in the transgenic knockout animal exhibiting decreased expression levels of the one or more genes as compared to a wild-type animal.

In another aspect, the invention provides a method to screen for therapeutic agents that modulate symptoms of schizophrenia by administering a candidate compound to the transgenic nonhuman animals disclosed above and determining the effect of the compound on symptoms associated with schizophrenia.

DETAILED DESCRIPTION OF PREFERRED EMBODIMENTS

In practicing the present invention, many conventional techniques in molecular biology are used. These techniques are well known and are explained in, for example, Current Protocols in Molecular Biology, Volumes I, II, and III, 1997 (F. M. Ausubel ed.); Sambrook et al., 1989, Molecular Cloning: A Laboratory Manual, Second Edition, Cold Spring Harbor Laboratory Press, Cold Spring Harbor, N.Y.; DNA Cloning: A Practical Approach, Volumes I and II, 1985 (D. N. Glover ed.); Oligonucleotide Synthesis, 1984 (M. L. Gait ed.); Nucleic Acid Hybridization, 1985, (Hames and Higgins); Transcription and Translation, 1984 (Hames and Higgins eds.); Animal Cell Culture, 1986 (R. I. Freshney ed.); Immobilized Cells and Enzymes, 1986 (IRL Press); Perbal, 1984, A Practical Guide to Molecular Cloning; the series, Methods in Enzymology (Academic Press, Inc.); Gene Transfer Vectors for Mammalian Cells, 1987 (J. H. Miller and M. P. Calos eds., Cold Spring Harbor Laboratory); and Methods in Enzymology Vol. 154 and Vol. 155 (Wu and Grossman, and Wu, eds., respectively).

As used herein and in the appended claims, the singular forms "a", "an", and "the" include plural reference unless the context clearly dictates otherwise. Thus, for example, reference to the "antibody" is a reference to one or more antibodies and equivalents thereof known to those skilled in the art, and so forth.

As used herein, the definition of a "schizophrenic disease or disorder" encompasses the characterization of this disease as described in the references cited above .

"Nucleic acid sequence", as used herein, refers to an oligonucleotide, nucleotide or polynucleotide, and fragments or portions thereof, and to DNA or RNA

of genomic or synthetic origin that may be single or double stranded, and represent the sense or antisense strand.

The term "antisense" as used herein, refers to nucleotide sequences which are complementary to a specific DNA or RNA sequence. The term "antisense strand" is used in reference to a nucleic acid strand that is complementary to the "sense' strand. Antisense molecules may be produced by any method, including synthesis by ligating the gene(s) of interest in a reverse orientation to a viral promoter which permits the synthesis of a complementary strand. Once introduced into a cell, this transcribed strand combines natural sequences produced by the cell to form duplexes. These duplexes then block either the further transcription or translation. The designation "negative " is sometimes used in reference to the antisense strand, and "positive" is sometimes used in reference to the sense strand.

As contemplated herein, antisense oligonucleotides, triple helix DNA, RNA aptamers, RNAi, ribozymes and double or single stranded RNA are directed to a nucleic acid sequence of a gene disclosed in Table 1 such that the nucleotide sequence of the gene chosen will produce gene-specific inhibition of gene expression. For example, knowledge of the target gene nucleotide sequence may be used to design an antisense molecule which gives strongest hybridization to the mRNA. Similarly, ribozymes can be synthesized to recognize specific nucleotide sequences and cleave them (Cech. J. Amer. Med Assn. 260:3030 (1988). Techniques for the design of such molecules for use in targeted inhibition of gene expression is well known to one of skill in the art.

As used herein, the term "antibody" refers to intact molecules as well as fragments thereof, such as Fa, $F(ab')_2$, and Fv, which are capable of binding the epitopic determinant. Antibodies that bind polypeptides of interest can be prepared using intact polypeptides or fragments containing small peptides of interest as the immunizing antigen. The polypeptides or peptides used to immunize an animal can be derived from the translation of RNA or synthesized chemically, and can be conjugated to a carrier protein, if desired. Commonly used carriers that are

chemically coupled to peptides include bovine serum albumin and thyroglobulin. The coupled peptide is then used to immunize an animal (e.g., a mouse, a rat or a rabbit).

The term "humanized antibody" as used herein, refers to antibody molecules in which amino acids have been replaced in the non-antigen binding regions in order to more closely resemble a human antibody, while still retaining the original binding ability.

A "therapeutically effective amount" is the amount of drug sufficient to treat and /or ameliorate the pathological effects of chronic pain, including but not limited to, hyperalgesia.

The term " therapeutic agent" as used herein describes any molecule, e.g. protein, carbohydrate, metal or organic compound, with the capability of affecting the molecular and clinical phenomena associated with schizophrenia. Generally a plurality of assay mixtures may be run in parallel with different agent concentrations to obtain a differential response to the various concentrations. Typically, one of these concentrations serves as a negative control, i.e. at zero concentration or below the level of detection.

"Subject" refers to any human or nonhuman organism.

The present disclosure is based on the surprising discovery that nineteen genes are associated with schizophrenia in affected individuals. More particularly, these genes are upregulated in the anterior cingulate of schizophrenic patients as compared to normal patients. The complete list of these genes is disclosed below in Table 1.

Table 1

GENE NAME	ABBREVIATION USED HEREIN
Small inducible cytokineA2	SCYA2
Growth arrest and DNA-damage-inducible beta	GADD45B

S100 calcium binding protein A8	S100A8
Cyclin-dependent kinase inhibitor 1A p21/Cip1	CDKN1A
Interleukin 1receptor-like 1	IL1RL1
Transglutaminase	TGM2
V-maf musculo aponeurotic fibrosarcoma oncogene homolog F	MAFF
Serine or cysteine proteinase inhibitor clade A member 3	SERPINA3
GRO1 oncogene melanoma growth stimulating activityalpha	GRO1
CD14 antigen	CD14
Tensin 2	KIAA1075
Chitinase 3-like 1, cartilage glycoprotein-39	CHI3L1
Serine or cysteine proteinase inhibitor clade H	SERPINH1
Metallothionein 1X	MT1X
KIAA0620 protein	KIAA0620
Tissue inhibitor of metalloproteinase 1	TIMP1
Nuclear mitotic apparatus protein 1	NUMA1
DNA-damage-inducibletranscript 3	DDIT3
Transducer of ERBB2	TOB2

Accordingly, methods for the diagnosis, screening and evaluation of schizophrenia are provided in accordance with the present invention. For example, assays for determination of increased levels of expression of these genes are provided. Moreover, nucleic acid molecules encoding these genes can be used as diagnostic hybridization probes or used to design primers for diagnostic PCR analysis for the identification of gene mutations, allelic variations and regulatory defects in these genes. As used herein, "diagnosis" is intended to generally apply to individuals while "screening" is generally applicable to populations or individuals. The invention also encompasses antibodies to the products of the genes disclosed in

Table 1 that can be used to decrease available plasma levels of these proteins, as well as nucleotide sequences that can be used to inhibit gene expression (e.g., antisense, RNAi and ribozyme molecules), and gene or regulatory sequence binding or replacement constructs designed to reduce or enhance gene expression (e.g., triple helix forming moieties or expression constructs that place the genes under the control of a strong promoter system).

The surprising expression characteristics of the genes disclosed in Table 1 were uncovered by examination of post mortem anterior cingulate samples from schizophrenic and normal subjects. Samples possessing high quality RNA were utilized for further study. Those skilled in the art are familiar with techniques which may be utilized to determine expression levels. For example, reverse transcriptase assays or DNA microarray analysis can be performed utilizing gene chip technology. Differentially expressed genes can be identified using a number of methods developed in accordance with established principles. Statistical significance of the expression differences between groups of samples may be determined utilizing the t-test, ANOVA or non-parametric tests. In accordance with the present invention, some genes were found to be upregulated in schizophrenic patients while others were found to be downregulated compared to baseline or normal levels. The terms "normal" and "baseline" are used interchangeably herein. Baseline levels are defined using conventional statistical techniques in connection with an analysis of a general population of non-schizophrenics. See, e.g., Example 1 herein. It should be understood, in general, that methods not otherwise specified herein are conducted in accordance with generally accepted principles known to those skilled in the art.

Quantitative rtPCR (Q-PCR) may be conducted on the same samples used for the expression level analysis described above. After conversion of RNA to cDNA using reverse transcriptase, although any conventional PCR technique can be utilized, a preferred technique may be based on the TaqMan® technique (Perkin Elmer Corp., Foster City, CA). In conventional PCR assays, oligonucleotide primers are designed complementary to the 5' and 3'ends of a DNA sequence of interest. During thermal cycling, DNA is heat denatured. The sample is then brought to

annealing and extension temperatures in which the primers bind their specific complements and are extended by the addition of nucleotide tri-phosphates by Taq polymerase. With repeated thermal cycling, the amount of template DNA is amplified. The presence of a dye, such as SybrGreen™, that fluoresces strongly when bound to DNA, allows the real time monitoring of total amount of DNA product in the tube. By measuring this signal, the amplified product can be quantified. The threshold cycle (C_T) at which the fluorescent signal is measurably different from the background noise is an accurate measure of the starting amount of cDNA in the tube and hence RNA in the sample. This method allows the quantitation of genes in a complex RNA by targeting specific DNAs. Of the genes initially identified by microarray analysis to be differentially expressed in schizophrenic patients, twenty two, decidual protein induced by progesterone (DEPP), csdA ,adrenomedullin as well as those disclosed herein in Table 1, were shown to be differentially regulated in the original set of RNA samples.

In one aspect, a method of screening for schizophrenia in a population is provided which includes determining, in members of the population, the magnitude of expression of a gene selected from those disclosed in Table 1in a sample and comparing the magnitude of expression to a baseline magnitude of expression of the gene, wherein increased gene expression indicates the presence of schizophrenia.

In another aspect, a method for diagnosing schizophrenia in a host is provided which includes determining the magnitude of expression of a gene consisting of those disclosed in Table 1 in a sample and comparing the magnitude of expression to a baseline magnitude of expression of the gene, wherein increased gene expression indicates the presence of schizophrenia. In either of the above screening or diagnosing aspects, the sample may be taken, for example, from the brain, spinal cord, lymphatic fluid, blood, urine or feces.

There are numerous techniques known to those with skill in the art to measure gene expression in a sample. For example, RNA from a cell type or tissue known, or suspected, to express a gene disclosed in Table 1 , such as brain, may be isolated

and tested utilizing hybridization or PCR techniques such as are described above. The isolated RNA can be derived directly from a biological sample from a patient.

In one embodiment of such a detection scheme, a cDNA molecule is synthesized from an RNA molecule of interest (e.g., by reverse transcription of the RNA molecule into cDNA). A sequence within the cDNA is then used as the template for a nucleic acid amplification reaction, such as a PCR amplification reaction, or the like. The nucleic acid reagents used as synthesis initiation reagents (e.g., primers) in the reverse transcription and nucleic acid amplification steps of this method are chosen from among the genes disclosed in Table 1. . Those skilled in the art are familiar with techniques for designing and obtaining suitable primers. See, e.g., Table 2 in Example 2 below. The preferred lengths of such nucleic acid reagents are at least 9-30 nucleotides. For detection of the amplified product, the nucleic acid amplification may be performed using radioactively or non-radioactively labeled nucleotides. Alternatively, enough amplified product may be made such that the product may be visualized by standard ethidium bromide staining or by utilizing any other suitable nucleic acid staining method.

Additionally, it is possible to perform such gene expression assays "in situ", i.e., directly upon tissue sections (fixed and/or frozen) of patient tissue obtained from biopsies or resections, such that no nucleic acid purification is necessary. Nucleic acid reagents such as those described above may be used as probes and/or primers for such in situ procedures. Alternatively, if a sufficient quantity of the appropriate cells can be obtained, standard Northern analysis can be performed to determine the level of mRNA expression of a gene disclosed in Table 1.

Regardless of the method used to quantify the expression of a gene or genes disclosed in Table 1, the level of expression in a subject of undefined etiology is compared to a known normal expression level. If the expression level of one, or more than one, of these genes is elevated above the normal or baseline level by about 25%, a diagnosis of schizophrenia may be made or confirmed. Determination of higher levels may be indicative of the severity of the disease.

As demonstrated by the Examples below, one technique for establishing baseline levels may involve real time quantitative PCR. Those skilled in the art are familiar with numerous techniques which may be utilized to test sample populations to obtain statistically sound results. For example, in carrying out this technique, a sample from a population of normal individuals is selected. The sample should be sufficiently diverse in terms of age, sex, social status, geographical distribution, previous drug and medical histories, etc. and of sufficient size to provide a meaningful statistical value. Thus, expression of a gene disclosed in Table 1 is measured in the sample of interest which defines distribution in the normal population. Baseline levels are then assigned. A set of diseased subjects is also assayed to determine validity of the test by comparing results of the diseased sample to those of the normal sample.

In accordance with the present invention, symptoms of schizophrenia associated with upregulation of a gene or genes disclosed in Table 1 may be ameliorated by decreasing the level of any one or more of these genes or gene product activity by using appropriately designed gene sequences in conjunction with well-known antisense, gene "knock-out," ribozyme, RNAi and/or triple helix methods to decrease the level of expression of any one or more genes disclosed in Table 1.

Among the compounds that may exhibit the ability to modulate the activity, expression or synthesis of genes disclosed in Table 1 including the ability to ameliorate the symptoms of schizophrenia associated with overexpression of any one or more of these genes, are antisense, ribozyme, RNAi and triple helix molecules. Such molecules may be designed to reduce or inhibit either unimpaired, or if appropriate, mutant target gene activity. Techniques for the production and use of such molecules are well known to those skilled in the art.

Antisense RNA and DNA molecules act to block the translation of mRNA by hybridizing to target mRNA and preventing protein translation. Antisense approaches involve the design of oligonucleotides that are complementary to a target gene

mRNA. The antisense oligonucleotides will bind to the complementary target gene mRNA transcripts and prevent translation. Absolute complementarity, although preferred, is not required.

A sequence "complementary" to a portion of an RNA, as referred to herein, means a sequence having sufficient complementarity to be able to hybridize with the RNA, forming a stable duplex; in the case of double-stranded antisense nucleic acids, a single strand of the duplex DNA may thus be tested, or triplex formation may be assayed. The ability to hybridize will depend on both the degree of complementarity and the length of the antisense nucleic acid. Generally, the longer the hybridizing nucleic acid, the more base mismatches with an RNA it may contain and still form a stable duplex (or triplex, as the case may be). One skilled in the art can ascertain a tolerable degree of mismatch by use of standard procedures to determine the melting point of the hybridized complex.

In one embodiment, oligonucleotides complementary to coding or non-coding regions of a gene disclosed in Table 1 could be used in an antisense approach to inhibit translation of the endogenous mRNA for any one or more of these genes. mRNA. Based upon the sequences presented herein, or upon allelic or homologous genomic and/or DNA sequences, one of skill in the art can easily choose and synthesize any of a number of appropriate antisense molecules for use in accordance with the present invention. Antisense nucleic acids should be at least six nucleotides in length, and are preferably oligonucleotides ranging from 6 to about 50 nucleotides in length. In certain preferred aspects the oligonucleotide length is from about 8 to about 30 nucleotides.

Suitable antisense oligonucleotides herein encompass modified oligonucleotides which may exhibit enhanced stability, targeting or which otherwise exhibit enhanced therapeutic effectiveness. Examples of modified oligonucleotides include those where (1) at least two nucleotides are covalently linked via a synthetic internucleoside linkage (i.e., a linkage other than a phosphodiester linkage between the 5' end of one nucleotide and the 3' end of another nucleotide) and/or (2) a

chemical group not normally associated with nucleic acids has been covalently attached to the oligonucleotide. Examples of synthetic internucleoside linkages are phosphorothioates, alkylphosphonates, phosphorodithioates, phosphate esters, alkylphosphonothioates, phosphoramidates, carbamates, phosphate triesters, acetamidates, peptides, and carboxymethyl esters. Modified oligonucleotides may also have covalently modified bases and/or sugars. For example, oligonucleotides having backbone sugars which are covalently attached to low molecular weight organic groups other than a hydroxyl group at the 3' position and other than a phosphate group at the 5' position. Thus modified oligonucleotides may include a 2'-O-alkylated ribose group. In addition, modified oligonucleotides may include sugars such as arabinose instead of ribose. Modified oligonucleotides also can include base analogs such as C-5 propyne modified bases.

Antisense oligonucleotides may be synthesized by standard techniques known in the art, e.g., by use of an automated DNA synthesizer (such as are commercially available from Biosearch, Applied Biosystems, etc.). As examples, phosphorothioate oligonucleotides may be synthesized by the method of Stein, et al. (1988, Nucl. Acids Res. 16, 3209), methylphosphonate oligonucleotides can be prepared by use of controlled pore glass polymer supports (Sarin, et al., 1988, Proc. Natl. Acad. Sci. U.S.A. 85, 7448-7451), etc.

While antisense nucleotides complementary to the target gene coding region sequence could be used, those complementary to the transcribed, untranslated region are most preferred. A preferred site is the region encompassing the translation initiation or termination codon of the open reading frame (ORF) of the gene. Those with skill in the art are well aware of various suitable initiation or termination codons in both eukaryotes and prokaryotes.

Antisense molecules may be delivered to cells that express the target gene in vivo. A number of methods have been developed for delivering antisense DNA or RNA to cells; e.g., antisense molecules can be injected directly into the tissue site, or modified antisense molecules, designed to target the desired cells (e.g., antisense

linked to peptides or antibodies that specifically bind receptors or antigens expressed on the target cell surface) can be administered systemically. A preferred technique involves constructing a vector which incorporates a strong promoter to provide high expression and good yield of antisense oligonucleotides at the target site. The use of such a construct to transfect target cells in the patient results in the transcription of sufficient amounts of single stranded RNAs that will form complementary base pairs with the endogenous target gene transcripts and thereby prevent translation of the target gene mRNA. For example, a vector can be introduced such that it is taken up by a cell and directs the transcription of an antisense RNA. Such a vector can remain episomal or become chromosomally integrated, as long as it can be transcribed to produce the desired antisense RNA. Such vectors can be constructed by recombinant DNA technology methods known to those in the art. Vectors can be, e.g., plasmid, viral, or others typically used for replication and expression in mammalian cells. It should be understood that expression of the sequence encoding the antisense RNA can be by any promoter known in the art to act in mammalian, preferably human cells. Such promoters can be inducible or constitutive. Any type of plasmid, cosmid, YAC, BAC or viral vector can be used to prepare the recombinant DNA construct which can be introduced directly into the tissue site. Alternatively, viral vectors can be used that selectively infect the desired tissue, in which case administration may be accomplished by another route (e.g., systemically).

Ribozyme molecules designed to catalytically cleave target gene mRNA transcripts can also be used to prevent or reduce translation of mRNA of any one or more genes diclosed in Table 1 herein and, therefore, expression of target gene product. (See, e.g., PCT International Publication W090/11364, published Oct. 4, 1990; Sarver, et: al., 1990, Science 247, 1222-1225). Ribozymes are enzymatic RNA molecules capable of catalyzing the specific cleavage of RNA. The mechanism of ribozyme action involves sequence specific hybridization of the ribozyme molecule to complementary target RNA, followed by an endonucleolytic cleavage event. The composition of ribozyme molecules must include one or more sequences complementary to the target gene mRNA, and must include the well known catalytic

sequence responsible for mRNA cleavage. For this sequence, see, e.g., U.S. Pat. No. 5,093,246, incorporated herein by reference.

Ribozymes that cleave mRNA at site specific recognition sequences can be used to destroy target gene mRNAs. For example, hammerhead ribozymes may be utilized to cleave mRNAs at locations dictated by flanking regions that form complementary base pairs with the target mRNA. The sole requirement is that the target mRNA have the following sequence of two bases: 5'-UG-3'. The construction and production of hammerhead ribozymes is well known in the art. Preferably, the ribozyme is engineered so that the cleavage recognition site is located near the 5' end of the target gene mRNA, i.e., to increase efficiency and minimize the intracellular accumulation of non-functional protein fragments. Suitable ribozymes also include RNA endoribonucleases such as the one that occurs naturally in Tetrahymena thermophila (known as the IVS, or L-19 IVS RNA). This type of ribozymes have an eight base pair active site which hybridizes to a target RNA sequence to effect cleavage of the target RNA.

As in the antisense approach, the ribozymes can be composed of modified oligonucleotides (e.g., for improved stability, targeting, etc.) and should be delivered to cells that express the target gene in vivo. A preferred method of delivery involves using a DNA construct "encoding" the ribozyme under the control of a strong constitutive promoter, so that transfected cells will produce sufficient quantities of the ribozyme to destroy endogenous gene messages and inhibit translation. Because ribozymes, unlike antisense molecules, are catalytic, a lower intracellular concentration is required for efficiency.

Alternatively, endogenous expression of any one of more genes disclosed in Table 1 can be reduced by targeting deoxyribonucleotide sequences complementary to the regulatory region of the target genes (i.e., the target gene promoter and/or enhancers) to form triple helical structures that prevent transcription of the target gene in target cells in the body. Nucleic acid molecules to be used in triplex helix formation for the inhibition of transcription should be single stranded and composed

of deoxynucleotides. The base composition of these oligonucleotides must be designed to promote triple helix formation via Hoogsteen base pairing rules, which generally require sizeable stretches of either purines or pyrimidines to be present on one strand of a duplex. Nucleotide sequences may be pyrimidine-based, which will result in TAT and CGC^+ triplets across the three associated strands of the resulting triple helix. The pyrimidine-rich molecules provide base complementarity to a purine-rich region of a single strand of the duplex in a parallel orientation to that strand. In addition, nucleic acid molecules may be chosen that are purine-rich, for example, contain a stretch of G residues. These molecules will form a triple helix with a DNA duplex that is rich in GC pairs, in which the majority of the purine residues are located on a single strand of the targeted duplex, resulting in GGC triplets across the three strands in the triplex.

Alternatively, the potential sequences that can be targeted for triple helix formation may be increased by creating a so-called "switchback" nucleic acid molecule. Switchback molecules are synthesized in an alternating 5'-3', 3'-5' manner, such that they base pair with first one strand of a duplex and then the other, eliminating the necessity for a sizeable stretch of either purines or pyrimidines to be present on one strand of a duplex.

RNA aptamers can also be introduced into or expressed in a cell to modify RNA abundance or activity. RNA aptamers are specific RNA ligands for proteins, such as for Tat and Rev RNA (Good et al., 1997, Gene Therapy 4: 45-54) that can specifically inhibit their translation. In addition, gene specific inhibition of gene expression may also be achieved using conventional double or single stranded RNA technologies. A description of such technology may be found in WO 99/32619 which is hereby incorporated by reference in its entirety. In addition, siRNA technology has also proven useful as a means to inhibit gene expression (Cullen, BR Nat. Immunol. 2002 Jul;3(7):597-9; J Martinez et al., Cell 2002 Sept. 6;110(5):563).

Anti-sense RNA and DNA, ribozyme, RNAi, RNA aptamer and triple helix molecules described herein may be prepared by any method known in the art for the

synthesis of DNA and RNA molecules, as discussed above. These include techniques for chemically synthesizing oligodeoxyribonucleotides and oligoribonucleotides well known in the art such as for example solid phase phosphoramidite chemical synthesis. Alternatively, RNA molecules may be generated by in vitro and in vivo transcription of DNA sequences encoding the antisense RNA molecule. Such DNA sequences may be incorporated into a wide variety of vectors that incorporate suitable RNA polymerase promoters such as the T7 or SP6 polymerase promoters. Alternatively, antisense cDNA constructs that synthesize antisense RNA constitutively or inducibly, depending on the promoter used, can be introduced stably into cell lines.

A method of modulating the activity of a protein encoded by a gene disclosed in Table 1 to treat schizophrenia is provided comprising exposing neutralizing antibodies to said proteins. By providing for controlled exposure to such antibodies, protein abundances/activities can be controllably modified. For example, antibodies to suitable epitopes on protein surfaces may decrease the abundance, and thereby indirectly decrease the activity, of the wild-type active form of a protein encoded by a gene disclosed in Table 1 by aggregating active forms into complexes with less or minimal activity as compared to the wild-type unaggregated wild-type form. Alternatively, , antibodies may directly decrease protein activity by, e.g., interacting directly with active sites or by blocking access of substrates to active site. In either case, antibodies can be raised against specific protein species and their effects screened. The effects of the antibodies can be assayed and suitable antibodies selected that lower the target protein species concentration and/or activity. Such assays involve introducing antibodies into a cell or surrounding media, and assaying the concentration of the wild-type amount or activities of the target protein by standard means (such as immunoassays) known in the art. The net activity of the wild-type form can be assayed by assay means appropriate to the known activity of the target protein.

Antibodies can be introduced into cells in numerous ways, including, for example, microinjection of antibodies into a cell (Morgan et al., 1988, Immunology

Today 9:84-86) or transforming hybridoma mRNA encoding a desired antibody into a cell (Burke et al., 1984, Cell 36:847-858). In a further technique, recombinant antibodies can be engineered and ectopically expressed in a wide variety of non-lymphoid cell types to bind to target proteins as well as to block target protein activities. Preferably, expression of the antibody is under control of a controllable promoter, such as the Tet promoter. A first step is the selection of a particular monoclonal antibody with appropriate specificity to the target protein. Then sequences encoding the variable regions of the selected antibody can be cloned into various engineered antibody formats, including, for example, whole antibody, Fab fragments, Fv fragments, single chain Fv fragments (VH and VL regions united by a peptide linker) ("ScFv" fragments), diabodies (two associated ScFv fragments with different specificities), and so forth. Intracellularly expressed antibodies of the various formats can be targeted into cellular compartments by expressing them as fusions with the various known intracellular leader sequences.

Methods for the production of antibodies capable of specifically recognizing one or more Table 1 gene product epitopes or or epitopes of conserved variants or peptide fragments of the proteins encoded by the genes disclosed in Table 1 are well known in the art. Such antibodies may include, but are not limited to, polyclonal antibodies, monoclonal antibodies (mAbs), humanized or chimeric antibodies, single chain antibodies, Fab fragments, $F(ab')_2$ fragments, fragments produced by a Fab expression library, anti-idiotypic (anti-Id) antibodies, and epitope-binding fragments of any of the above.

Such antibodies may also be used, for example, in the detection of a Table 1 gene product in an biological sample and may, therefore, be utilized as part of a diagnostic or prognostic technique whereby patients may be tested for abnormal levels of any one or more of said gene products, and/or for the presence of abnormal forms of such gene products. Such antibodies may also be utilized in conjunction with, for example, compound screening schemes, for the evaluation of the effect of test compounds on any one or more of said gene product levels and/or activity.

For the production of antibodies against any one or more of the gene products disclosed herein various host animals may be immunized by injection with a Table 1 gene product, or a portion thereof. Such host animals may include, but are not limited to, rabbits, mice, goats, chickens and rats, to name but a few. Various adjuvants may be used to increase the immunological response, depending on the host species, including but not limited to Freund's (complete and incomplete), mineral gels such as aluminum hydroxide, surface active substances such as lysolecithin, pluronic polyols, polyanions, peptides, oil emulsions, keyhole limpet hemocyanin, dinitrophenol, and potentially useful human adjuvants such as BCG (bacille Calmette-Guerin) and Corynebacterium parvum.

Polyclonal antibodies are heterogeneous populations of antibody molecules derived from the sera of animals immunized with an antigen, such as a Table 1 gene product, or an antigenic functional derivative thereof. For the production of polyclonal antibodies, host animals such as these described above, may be immunized by injection with a Table 1 gene product supplemented with adjuvants as described above.

Monoclonal antibodies, which are homogeneous populations of antibodies to a particular antigen, may be obtained by any technique that provides for the production of antibody molecules by continuous cell lines in culture. These include, but are not limited to, the hybridoma technique of Kohler and Milstein, (1975, Nature 256, 495-497; and U.S. Pat. No. 4,376,110), the human B-cell hybridoma technique (Kosbor et al., 1983, Immunology Today 4, 72; Cole et al., 1983, Proc. Natl. Acad. Sci. USA 80, 2026-2030), and the EBV-hybridoma technique (Cole et al., 1985, Monoclonal Antibodies And Cancer Therapy, Alan R. Liss, Inc., pp. 77-96). Such antibodies may be of any immunoglobulin class including IgG, IgM, IgE, IgA, IgD and any subclass thereof. The hybridoma producing the mAb may be cultivated in vitro or in vivo.

In addition, techniques developed for the production of "chimeric antibodies" (Morrison, et al., 1984, Proc. Natl. Acad. Sci., 81, 6851-6855; Neuberger, et al., 1984,

Nature 312, 604-608; Takeda, et al., 1985, Nature, 314, 452-454) by splicing the genes from a mouse antibody molecule of appropriate antigen specificity together with genes from a human antibody molecule of appropriate biological activity can be used. A chimeric antibody is a molecule in which different portions are derived from different animal species, such as those having a variable region derived from a murine mAb and a human immunoglobulin constant region. Techniques have also been developed for the production of humanized antibodies. (See, e.g., Queen, U.S. Pat. No. 5,585,089,). An immunoglobulin light or heavy chain variable region consists of a "framework" region interrupted by three hypervariable regions, referred to as complementarity determining regions (CDRs). The extent of the framework region and CDRs have been precisely defined (see, e.g., "Sequences of Proteins of Immunological Interest", Kabat, E. et al., U.S. Department of Health and Human Services (1983)). Briefly, humanized antibodies are antibody molecules from non-human species having one or more CDRs from the non-human species and a framework region from a human immunoglobulin molecule. Alternatively, techniques described for the production of single chain antibodies (U.S. Pat. No. 4,946,778; Bird, 1988, Science 242, 423-426; Huston, et al., 1988, Proc. Natl. Acad. Sci. USA 85, 5879-5883; and Ward, et al., 1989, Nature 334, 544-546) can be adapted to produce single chain antibodies against any one or more of the proteins disclosed herein. Single chain antibodies are formed by linking the heavy and light chain fragments of the Fv region via an amino acid bridge, resulting in a single chain polypeptide.

Antibody fragments that recognize specific epitopes may be generated by known techniques. For example, such fragments include but are not limited to: the $F(ab')_2$ fragments, which can be produced by pepsin digestion of the antibody molecule and the Fab fragments, which can be generated by reducing the disulfide bridges of the $F(ab')_2$ fragments. Alternatively, Fab expression libraries may be constructed (Huse, et al., 1989, Science, 246, 1275-1281) to allow rapid and easy identification of monoclonal Fab fragments with the desired specificity.

Antibodies, or fragments of antibodies, such as those described, above, may be used to quantitatively or qualitatively detect the presence of any one or more

Table 1 gene product or conserved variants or peptide fragments thereof. This can be accomplished, for example, by immunofluorescence techniques employing a fluorescently labeled antibody coupled with light microscopic, flow cytometric, or fluorometric detection.

The antibodies (or fragments thereof) useful in the present invention may be employed histologically, as in immunofluorescence or immunoelectron microscopy, for in situ detection of any one or more Table 1 gene product, conserved variants or peptide fragments thereof. In situ detection may be accomplished by removing a histological specimen from a patient, and applying thereto a labeled antibody that binds to a polypeptide encoded by a gene disclosed in Table 1.. The antibody (or fragment) is preferably applied by overlaying the labeled antibody (or fragment) onto a biological sample. Through the use of such a procedure, it is possible to determine not only the presence of any one or more of said polypeptide, conserved variant or peptide fragment, but also its distribution in the examined tissue. Using the present invention, those of ordinary skill will readily recognize that any of a wide variety of histological methods (such as staining procedures) can be modified in order to achieve in situ detection the product of a gene disclosed in Table 1

Immunoassays for a product of a gene disclosed in Table 1 conserved variants, or peptide fragments thereof will typically comprise incubating a sample, such as a biological fluid, a tissue extract, freshly harvested cells, or lysates of cells in the presence of a detectably labeled antibody capable of identifying said gene product, conserved variant or peptide fragments thereo, and detecting the bound antibody by any of a number of techniques well-known in the art. The biological sample may be brought in contact with and immobilized onto a solid phase support or carrier, such as nitrocellulose, that is capable of immobilizing cells, cell particles or soluble proteins. The support may then be washed with suitable buffers followed by treatment with the detectably labeled protein appropriate specific antibodies. The solid phase support may then be washed with the buffer a second time to remove unbound antibody. The amount of bound label on the solid support may then be detected by conventional means.

One of the ways in which specific antibodies can be detectably labeled is by linking the same to an enzyme, such as for use in an enzyme immunoassay (EIA). The enzyme, which is bound to the antibody, will react with an appropriate substrate, preferably a chromogenic substrate, in such a manner as to produce a chemical moiety that can be detected, for example, by spectrophotometric, fluorimetric or by visual means. Enzymes that can be used to detectably label the antibody are well known. The detection can be accomplished by colorimetric methods that employ a chromogenic substrate for the enzyme. Detection may also be accomplished by visual comparison of the extent of enzymatic reaction of a substrate in comparison with similarly prepared standards. Detection may also be accomplished using any of a variety of other immunoassays. For example, by radioactively labeling the antibodies or antibody fragments, it is possible to detect any one or more proteins encoded by the genes disclosed in Table 1 through the use of a radioimmunoassay (RIA). The radioactive isotope can be detected by such means as the use of a gamma counter or a scintillation counter or by autoradiography. It is also possible to label the antibody with a fluorescent compound. When the fluorescently labeled antibody is exposed to light of the proper wavelength, its presence can then be detected due to fluorescence. Among the most commonly used fluorescent labeling compounds are green fluorescent protein, fluorescein isothiocyanate, rhodamine, phycoerythrin, phycocyanin, allophycocyanin, o-phthaldehyde and fluorescamine. The antibody can also be detectably labeled using fluorescence emitting metals such as ^{152}Eu, or others of the lanthanide series. These metals can be attached to the antibody using such metal chelating groups as diethylenetriaminepentacetic acid (DTPA) or ethylenediaminetetraacetic acid (EDTA). The antibody also can be detectably labeled by coupling it to a chemiluminescent compound. The presence of the chemiluminescent-tagged antibody is then determined by detecting the presence of luminescence that arises during the course of a chemical reaction. Examples of particularly useful chemiluminescent labeling compounds are luminol, isoluminol, theromatic acridinium ester, imidazole, acridinium salt and oxalate ester. Likewise, a bioluminescent compound may be used to label the antibody of the present invention. Bioluminescence is a type of chemiluminescence found in biological systems in

which a catalytic protein increases the efficiency of the chemiluminescent reaction. The presence of a bioluminescent protein is determined by detecting the presence of luminescence. Important bioluminescent compounds for purposes of labeling include luciferin, luciferase and aequorin.

The present invention contemplates production of animal models that have abnormal expression levels of any one or more genes disclosed in Table 1 to study the effects of increased or decreased levels of these proteins on such animals. Such animals provide test subjects for determining the effects of therapeutic or potentially therapeutic compounds on schizophrenia. Accordingly, Table 1gene products can be expressed in transgenic animals. Animals of any species, including, but not limited to, mice, rats, rabbits, guinea pigs, pigs, mini-pigs, goats, sheep, and non-human primates, e.g., baboons, monkeys, and chimpanzees may be used to generate these transgenic animals. The term "transgenic," as used herein, refers to animals expressing any one or more Table 1 gene sequence from a different species (e.g., mice expressing human gene sequences) , as well as animals that have been genetically engineered to overexpress endogenous (i.e., same species) gene sequences or animals that have been genetically engineered to no longer express endogenous gene sequences (i.e., "knockout" animals), and their progeny.

Any technique known in the art may be used to introduce genes into animals to produce the founder lines of transgenic animals. Such techniques include, but are not limited to, pronuclear microinjection (Hoppe and Wagner, 1989, U.S. Pat. No. 4,873,191); retrovirus mediated gene transfer into germ lines (Van der Putten, et al., 1985, Proc. Natl. Acad. Sci., USA 82, 6148-6152); gene targeting in embryonic stem cells (Thompson, et al., 1989, Cell 56, 313-321); electroporation of embryos (Lo, 1983, Mol. Cell. Biol. 3, 1803-1814); and sperm-mediated gene transfer (Lavitrano et al., 1989, Cell 57, 717-723) (For a review of such techniques, see Gordon, 1989, Transgenic Animals, Intl. Rev. Cytol. 115, 171-229). Any technique known in the art may be used to produce transgenic animal clones containing a transgene, for example, nuclear transfer into enucleated oocytes of nuclei from cultured embryonic,

fetal or adult cells induced to quiescence (Campbell, et al., 1996, Nature 380, 64-66; Wilmut, et al., 1997,Nature 385, 810-813).

The present invention provides for transgenic animals that carry a Table 1 transgene in all their cells, as well as animals that carry the transgene in some, but not all their cells, i.e., mosaic animals. The transgene may be integrated as a single transgene or in concatamers, e.g., head-to-head tandems or head-to-tail tandems. The transgene may also be selectively introduced into and activated in a particular cell type by following, for example, the teaching of Lasko et al. (Lasko, et al., 1992, Proc. Natl. Acad. Sci. USA 89, 6232-6236). The regulatory sequences required for such a cell-type specific activation will depend upon the particular cell type of interest, and will be apparent to those of skill in the art. When it is desired that the transgene be integrated into the chromosomal site of the endogenous gene, gene targeting is preferred. Briefly, when such a technique is to be utilized, vectors containing some nucleotide sequences homologous to the endogenous gene are designed for the purpose of integrating, via homologous recombination with chromosomal sequences, into and disrupting the function of the nucleotide sequence of the endogenous gene. The transgene may also be selectively introduced into a particular cell type, thus inactivating the endogenous gene in only that cell type, by following, for example, the teaching of Gu, et al., 1994, Science 265, 103-106. The regulatory sequences required for such a cell-type specific inactivation will depend upon the particular cell type of interest, and will be apparent to those of skill in the art.

As mentioned above, transgenic knockout animals are also provided herein. In such transgenic animals expression of any one or more genes disclosed in Table 1 is undetectable or insignificant. Any technique known in the art may be used to produce such transgenic knockout animals. This may be achieved by a variety of mechanisms, e.g., alteration of any or all of the Table 1 genes by, e.g., introduction of a disruption of the appropriate coding sequences, e.g., insertion of one or more stop codons, insertion of a DNA fragment, etc., deletion of regulatory or coding sequence, substitution of stop codons for coding sequence, etc. The transgenic animals may be either homozygous or heterozygous for the alteration. A functional

knock-out may also be achieved by the introduction of an anti-sense construct that blocks expression of the native genes. Knockouts also include conditional knockouts such as where alteration of the target gene occurs upon exposure of the animal to a substance that promotes target gene alteration, introduction of an enzyme that promotes recombination at the target gene site, or other method for directing the target gene alteration postnatally.

Once transgenic animals have been generated, the expression of the recombinant gene may be assayed utilizing standard techniques. Initial screening may be accomplished by Southern blot analysis or PCR techniques to analyze animal tissues to assay whether integration of the transgene has taken place. The level of mRNA expression of the transgene in the tissues of the transgenic animals may also be assessed using techniques described above and those that include but are not limited to Northern blot analysis of tissue samples obtained from the animal, in situ hybridization analysis, and RT-PCR (reverse transcriptase PCR). Samples of gene-expressing tissue, may also be evaluated immunocytochemically using antibodies specific for the transgene product of interest.

Through use of the subject transgenic animals or cells derived therefrom, one can identify ligands or substrates that modulate phenomena associated with schizophrenia, e.g., behavioral phenomena. A wide variety of assays may be used for this purpose, including behavioral studies, determination of the localization of drugs after administration and the like. Depending on the particular assay, whole animals may be used, or cells derived therefrom. Cells may be freshly isolated from an animal, or may be immortalized in culture. Cells of particular interest are derived from neural tissue.

Candidate therapeutic agents encompass numerous chemical classes, though typically they are organic molecules, preferably small organic compounds having a molecular weight of more than 50 and less than about 2,500 daltons. Candidate agents comprise functional groups necessary for structural interaction with proteins, particularly hydrogen bonding, and typically include at least an amine,

carbonyl, hydroxyl or carboxyl group, preferably at least two of the functional chemical groups. The candidate therapeutic agents often comprise cyclical carbon or heterocyclic structures and/or aromatic or polyaromatic structures substituted with one or more of the above functional groups. Candidate therapeutic agents are also found among biomolecules including, but not limited to: peptides, saccharides, fatty acids, steroids, purines, pyrimidines, derivatives, structural analogs or combinations thereof.

As mentioned above, antibodies specific for proteins encoded by the genes disclosed in Table 1 may be used in screening immunoassays, particularly to detect the level of such gene product in a cell or sample. The number of cells in a sample will generally be at least about 10^3, usually at least 10^4 more usually at least about 10^5. The cells may be dissociated, in the case of solid tissues, or tissue sections may be analyzed. Alternatively a lysate of the cells may be prepared. For example, detection may utilize staining of cells or histological sections, performed in accordance with conventional methods. The antibodies of interest are added to the cell sample, and incubated for a period of time sufficient to allow binding to the epitope, usually at least about 10 minutes. The antibody may be labeled with radioisotopes, enzymes, fluorescers, chemiluminescers, or other labels for direct detection. Alternatively, a second stage antibody or reagent is used to amplify the signal. Such reagents are well known in the art. For example, the primary antibody may be conjugated to biotin, with horseradish peroxidase-conjugated avidin added as a second stage reagent. Final detection uses a substrate that undergoes a color change in the presence of the peroxidase. The absence or presence of antibody binding may be determined by various methods, including flow cytometry of dissociated cells, microscopy, radiography, scintillation counting, etc.

A number of assays are known in the art for determining the effect of a drug on animal behavior and other phenomena associated with schizophrenia. Some examples are provided, although it will be understood by one of skill in the art that many other assays may also be used. The subject animals may be used by themselves, or in combination with control animals.

The screen using the transgenic animals of the invention can employ any phenomena associated with schizophrenia that can be readily assessed in an animal model. The screening for schizophrenia can include assessment of phenomena including, but not limited to: 1) analysis of molecular markers (e.g., levels of expression of any one or more Table gene products in brain tissue; presence/absence in brain tissue of various Table 1 gene splice variants; 2) assessment of behavioral symptoms associated with memory and learning; and 3) detection of neurodegeneration. Preferably, the screen will include control values (e.g., the level of production of a Table 1 gene product in the test animal in the absence of test compound(s)). Test substances which are considered positive, i.e., likely to be beneficial in the treatment of schizophrenia, will be those which have a substantial effect upon a schizophrenia associated phenomenon (e.g., test agents that are able to normalize erratic or abnormal behavior or that reduce the level of production of a Table 1 gene product to within the normal range).

The present invention also encompasses the use of cell-based assays or cell-lysate assays (e.g., in vitro transcription or translation assays) to screen for compounds or compositions that modulate the expression of any one or more genes disclosed in Table 1. To this end, constructs containing a reporter sequence linked to a regulatory element of a gene disclosed in Table 1 can be used in engineered cells, or in cell lysate extracts, to screen for compounds that modulate the expression of the reporter gene product at the level of transcription. For example, such assays could be used to identify compounds that modulate the expression or activity of transcription factors involved in expression of any one or more of the genes disclosed in Table 1 , or to test the activity of triple helix polynucleotides. Alternatively, engineered cells or translation extracts can be used to screen for compounds (including antisense and ribozyme constructs) that modulate the translation of Table 1 gene mRNA transcripts, and therefore, affect expression of these gene products. Thus, regulatory regions such as a promoter are operatively linked to a gene encoding a reporter molecule such as green fluorescent protein (GFP), luciferase and the like, to create a reporter construct which is regulated by appropriate regulatory

sequences for a gene disclosed in Table 1. The gene construct is then transfected into a desired cell such as a neuronal cell. The baseline expression levels of the reporter molecule are then calculated using conventional methods. The cell is then exposed to a test compound and the level of expression of the reporter molecule is determined and compared to the baseline levels. A compound which reduces the amount of reporter expression is a candidate for the treatment of schizophrenia. A second screening procedure may then be instituted to determine whether the compound affects the level of expression of any one or more genes disclosed in Table 1 by measuring the amount of RNA or protein from the native gene(s). Construction of neuronal cells incorporating a reporter gene for determining the effect of compounds on expression is known, e.g., see, Asselbergs et al., Nucleic Acids Res 27:1826-33(1998), incorporated herein by reference.

Antisense compounds, ribozymes, RNAi, RNA aptamers, antibodies and other geneknockout devices or modulators (collectively referred to for convenienceas the "modulators") described herein may be admixed, encapsulated, conjugated or otherwise associated with other molecules, molecular structures or mixtures of compounds, as for example, liposomes, receptor targeted molecules, oral, rectal, topical, or other formulations, for assisting in uptake, distribution and/or absorption. Those skilled in the art are familiar with a myriad of techniques to produce such devices.

It is contemplated that the modulators may encompass any pharmaceutically acceptable salts, esters, or salts of such esters, or any other compound which, upon administration to an animal including a human, is capable of providing (directly or indirectly) the biologically active metabolite or residue thereof. Accordingly, for example, the disclosure is also drawn to prodrugs and pharmaceutically acceptable salts of the compounds of the invention, pharmaceutically acceptable salts of such prodrugs, and other bioequivalents. The term "pharmaceutically acceptable salts" refers to physiologically and pharmaceutically acceptable salts of the compounds of the invention: i.e., salts that retain the desired biological activity of the parent compound and do not impart undue toxicological effects thereto. Such compounds

may be prepared according to conventional methods by one of skill in the art. (Berge et al., "Pharmaceutical Salts," J. of Pharma Sci., 1977, 66, 1-19). The term "prodrug" indicates a therapeutic agent that is prepared in an inactive form that is converted to an active form (i.e., drug) within the body or cells thereof by the action of endogenous enzymes or other chemicals and/or conditions. In particular, prodrug versions of the oligonucleotides may be prepared as SATE [(S-acetyl-2-thioethyl)phosphate] derivatives according to the methods disclosed in WO 93/24510 to Gosselin et al., or in WO 94/26764 to Imbach et al.

The modulators herein can be utilized for diagnostics, therapeutics, prophylaxis and as research reagents and kits. For therapeutics, an animal, preferably a human, suspected of having a schizophrenic disease or disorder which can be treated by modulating the expression of one or more genes disclosed in Table 1 , is treated by administering modulators in accordance with this invention. The modulators can be utilized in pharmaceutical compositions by adding an effective amount of one or more modulators to a suitable pharmaceutically acceptable diluent or carrier. Those skilled in the art are familiar with numerous techniques and formulations utilized to compound pharmaceutical compositions. The pharmaceutical compositions of the present invention may be administered in a number of ways depending upon whether local or systemic treatment is desired and upon the area to be treated. Administration may be topical (including ophthalmic and to mucous membranes including vaginal and rectal delivery), pulmonary, e.g., by inhalation or insufflation of liquids, powders or aerosols, including by nebulizer; intratracheal, intranasal, enteral, epidermal and transdermal), oral, sublingual, buccal or parenteral. Parenteral administration includes intravenous, intraarterial, subcutaneous, intraperitoneal or intramuscular injection or infusion; intramedullary or intracranial, e.g., intrathecal or intraventricular, administration. Oligonucleotides with at least one 2'-O-methoxyethyl modification may be useful for oral administration.

Pharmaceutical compositions for topical administration may include transdermal patches, ointments, lotions, creams, gels, drops, suppositories, sprays, liquids and powders. Conventional pharmaceutical carriers, aqueous, powder or oily

bases, thickeners and the like may be necessary or desirable. Compositions and formulations for oral administration include powders or granules, suspensions or solutions in water or non-aqueous media, capsules, sachets, troches or tablets. Thickeners, flavoring agents, diluents, emulsifiers, dispersing aids or binders may be desirable. Compositions for parenteral, intrathecal or intraventricular administration may include sterile aqueous solutions that may also contain buffers, diluents and other suitable additives such as, but not limited to, penetration enhancers, carrier compounds and other pharmaceutically acceptable carriers or excipients. Pharmaceutical compositions of the present invention include, but are not limited to, solutions, emulsions, suspensions, foams and liposome-containing formulations. These compositions may be generated from a variety of components that include, but are not limited to, preformed liquids, self-emulsifying solids and self-emulsifying semisolids, according to conventional methods, by one of skill in the art.

The pharmaceutical formulations of the present invention, which may conveniently be presented in unit dosage form, may be prepared according to conventional techniques well known in the pharmaceutical industry. Such techniques include the step of bringing into association the active ingredients with the pharmaceutical carrier(s) or excipient(s). In general, the formulations are prepared by uniformly and intimately bringing into association the active ingredients with liquid carriers or finely divided solid carriers or both, and then, if necessary, shaping the product. Further details on techniques for formulation and administration of numerous dosage forms may be found in the latest edition of Remington's Pharmaceutical Sciences (Maack Publishing Co., Easton, Pa.). The compositions may be administered alone or in combination with at least one other agent, such as stabilizing compound, which may be administered in any sterile, biocompatible pharmaceutical carrier, including, but not limited to, saline, buffered saline, dextrose, and water. The compositions may be administered to a patient alone, or in combination with other agents, drugs or hormones.

Pharmaceutical formulations suitable for parenteral administration may be formulated in aqueous solutions, preferably in physiologically compatible buffers such

as Hanks' solution, Ringer's solution, or physiologically buffered saline. Aqueous injection suspensions may contain substances that increase the viscosity of the suspension, such as sodium carboxymethyl cellulose, sorbitol, or dextran. Additionally, suspensions of the active compounds may be prepared as appropriate oily injection suspensions. Suitable lipophilic solvents or vehicles include fatty oils such as sesame oil, or synthetic fatty acid esters, such as ethyl oleate or triglycerides, or liposomes. Non-lipid polycationic amino polymers may also be used for delivery. Optionally, the suspension may also contain suitable stabilizers or agents that increase the solubility of the compounds to allow for the preparation of highly concentrated solutions. For topical or nasal administration, penetrants appropriate to the particular barrier to be permeated are used in the formulation. Such penetrants are generally known in the art.

Pharmaceutical compositions suitable for use in the invention include compositions wherein the modulators are contained in an effective amount to achieve the intended purpose. The determination of an effective dose is well within the capability of those skilled in the art. For any compound, the therapeutically effective dose can be estimated initially either in cell culture assays, e.g., of neoplastic cells, or in animal models, usually mice, rabbits, dogs, or pigs. The animal model may also be used to determine the appropriate concentration range and route of administration. Such information can then be used to determine useful doses and routes for administration in humans. A therapeutically effective dose refers to that amount of active ingredient, which ameliorates, partially or completely, the symptoms or condition. Therapeutic efficacy and toxicity may be determined by standard pharmaceutical procedures in cell cultures or experimental animals, e.g., ED50 (the dose therapeutically effective in 50% of the population) and LD50 (the dose lethal to 50% of the population). The dose ratio between toxic and therapeutic effects is the therapeutic index, and it can be expressed as the ratio, LD50/ED50. Pharmaceutical compositions that exhibit large therapeutic indices are preferred. The data obtained from cell culture assays and animal studies is used in formulating a range of dosage for human use. The dosage contained in such compositions is preferably within a range of circulating concentrations that include the ED50 with little or no toxicity. The

dosage varies within this range depending upon the dosage form employed, sensitivity of the patient, and the route of administration.

The exact dosage will be determined by the practitioner in light of factors related to the subject that require treatment. Dosage and administration are adjusted to provide sufficient levels of the modulators to maintain the desired effect. Factors which may be taken into account include the severity of the disease state, general health of the subject, age, weight, and gender of the subject, diet, time and frequency of administration, drug combination(s), reaction sensitivities, and tolerance/response to therapy. Long-acting pharmaceutical compositions may be administered every 3 to 4 days, every week, or once every two weeks depending on half-life and clearance rate of the particular formulation. Normal dosage amounts may vary from 0.1 to 100,000 micrograms, up to a total dose of about 1 g per kilogram, depending upon the route of administration. Guidance as to particular dosages and methods of delivery is provided in the literature and generally available to practitioners in the art. Those skilled in the art will employ different formulations for nucleotides than for proteins or their inhibitors. Similarly, delivery of polynucleotides or polypeptides will be specific to particular cells, conditions, locations, etc.

All references cited herein are incorporated by reference in their entireties. The following examples are included for purposes of illustration and should not be construed as limiting the present invention.

EXAMPLE 1

DNA Microarray Analysis

Human anterior cingulate samples are obtained from 20 normal and 20 schizophrenic deceased subjects (Maryland Psychiatric Research Clinic, Baltimore, Maryland). Good quality RNA was obtained from 19 normal ("N") and 18 schizophrenic ("S") samples.

The microarray analysis is performed essentially as follows. Briefly, 5μg or less total RNA is used to synthesize cDNA which is then used as a template to generate biotinylated cRNA. 15 to 30 μg labeled RNA is obtained and hybridized to Affymetrix (Santa Clara, CA) Human Genome U95Av2 Arrays of the GeneChip® Human Genome U95 Set (HG-U95Av2 contains ≈12,000 sequences of full length genes) in accordance with the protocols found in the GeneChip® technical manual. Each sample is profiled in duplicate. After sample hybridization, microarrays are washed and scanned with a laser scanner.

The images obtained are used to generate absolute text files for analysis using Affymetrix GeneChip® Gene Expression Analysis Algorithms version 4. Differentially expressed genes between the normal and schizophrenic derived samples are ranked using a pattern recognition algorithm developed in accordance with established principles which generated a score for each gene being compared. The following three conditions are required for a score (equal to the mean fold change) to be generated: (1) t-test p-value<0.5%; (2) average fold-change>1.5; (3) maximum mean AvgDiff (expression levels on an Affymetrix chip)>200. If one or more of the above conditions is not met by a gene in comparison, the score assigned is zero. Results indicate that several genes are found to be differentially expressed in schizophrenic patients when compared to normal (see Table 2 below).

EXAMPLE 2

Real Time Quantitative PCR Confirmation Of Differentially Regulated Genes

Probe pairs for real time quantitative PCR (Q-PCR) are designed for the 56 altered genes identified in Example 1. Affymetrix provides a file of sequences from which the probes on the chip are derived. From this file, the sequences corresponding to these 56 altered genes are obtained, and the probe pairs are prepared. Where a good pair of primers cannot be obtained from Affymetrix

sequence, a longer sequence can be obtained from Ref Seq. (See Pruitt KD, Maglott DR Nucleic Acids Res 2001 Jan 1;29(1):137-140;.Pruitt KD, et al. Trends Genet. 2000 Jan;16(1):44-47) with a good BLAST score against the Affymetrix sequence and the primers are designed from that sequence. The sequences of the probe pairs and the best RefSeq or Genbank hits are are presented in Table 2. Most were detected as differentially expressed in schizophrenic patients compared to normal by Affymetrix GeneChips®. ACTB and GAPD were included as controls.

Table 2

Oligopair	PCR Primer Sequences		RefSeq or GenBank IDs	GeneName
SZ1-29	CACCCAGCAGAGCAG TGTGA (Seq ID No 1)	TTTTGCTTTATTTCT GAATGGTCATCT (Seq ID No 59)	NM_003651	Cold shock domain protein A (CSDA)
SZ1-25	GAGTCTGAAGGACCC TAGTTCCTAGA (Seq ID No 2)	TCTGTCCCTTCACC TCTGATCA (Seq ID No 60)	NM_007021	Decidual protein induced by progesterone (DEPP)
SZ1-11	TCGCCCACAAACTGA TTTCTC (Seq ID No 3)	ACGCATTGCACTTT TCCTCTTT (Seq ID No 61)	NM_001124	Adrenomedullin (ADM)
JSZ9	GTGCCTGTAGTGACT GACAAGCA (Seq ID No 4)	AGGCCCCGGGTCT AGGA (Seq ID No 62)	NM_002673	Plexin B1 (PLXNB1)
JSZ8	TTCTGACAACTGGTG GCAGATT (Seq ID No 5)	TTGGACCCAGACG GGAAA (Seq ID No 63)	NM_002509	NK2 transcription factor homolog B (NKX2B)
JSZ7	GCCTCCCAGTGCAAA TCCT (Seq ID No 6)	CAGGGAGAAGAAC TGGGAGTTAACT (Seq ID No 64)	NM_013279	Chromosome 11 ORF 9 (C11orf9)
JSZ6	GACCTGTTGTAATTG CTCCTCATGT (Seq ID No 7)	ACGGCAAGGTATC GACAGGAT (Seq ID No 65)	AF305057	RTS gene (RTS)
JSZ5A	TATTAACAGGATAAC CCTTGAATGTAGCA (Seq ID No 8)	CCTCGGCCCTGGT CGTT (Seq ID No 66)	NM_004636	Immunoglobulin domain Ig secreted semaphorin3 (SEMA3B)
JSZ57	ACATGCCGTTGCTCA AAGCT (Seq ID No 9)	GCCATCAACTTCAA TTTCCTTTTC (Seq ID No 67)	NM_000784	Cytochrome P450 subfamily XXVIIA (CYP27A1)
JSZ56B	CAAGCAGAAGTGGGT TCAGGAT (Seq ID No 10)	TTAGCTGCAGATTC TTGGGTTGT (Seq ID No 68)	NM_002982	Small inducible cytokineA2 (SCYA2)
JSZ55B	GGACTCGATTCTGCC CTTCA (Seq ID No 11)	ACAATGGGCTCGAC TTAGCATAA (Seq ID No 69)	NM_004123	Gastric inhibitorypolypeptide (GIP)
JSZ54A	AAGGGATTCGGCCCA ATAAT (Seq ID No 12)	CAGAGACCAAGAA GGTCAAGATGTACT (Seq ID No 70)	NM_001686	ATP synthaseH+transporting mitochondrial F1 complex beta polypeptide(ATP5B)
JSZ53C	TCAATCCTGCATCCC CCATA (Seq ID No 13)	ACAGCCACCAGTGA GCTTCCT (Seq ID No 71)	NM_001511	GRO1 oncogene melanoma growth stimulating activityalpha (GRO1)
JSZ52A	ATGATCCTATTCTGTG TTAGCTCCAAT (Seq ID No 14)	TTCTTAAGGCTGTA ATTTATGCACAGTT (Seq ID No 72)	NM_001249	Ectonucleoside triphosphate diphosphohydrolase 5 (ENTPD5)
JSZ51	GACCCACCAGTGCCT TCTGT (Seq ID No 15)	CTCCCCACTTTGGG CACTTA (Seq ID No 73)	NM_002391	Midkine neurite growth-promoting factor 2 (MDK)
JSZ50	CTGCCTTTTCCTGCG	GACAGAGAGCCGC	NM_000591	CD14 antigen (CD14)

	AACA (Seq ID No 16)	CATCAGT (Seq ID No 74)		
JSZ49	ACAAGCTCAGAGCCC ACATCA (Seq ID No 17)	ATTCCTAAGGGAGG GTGCTTTCT (Seq ID No 75)	NM_015319	Tensin 2 (KIAA1075)
JSZ48	AGGGCACCACGCAG ACAT (Seq ID No 18)	CCTGGACAAGTTTG AAGGACAGA (Seq ID No 76)	BC036944	EST clone IMAGE:5395238
JSZ46	TGGAGTGTCGGATCC TGTGA (Seq ID No 19)	CTCCCACAAGAATG ATGATGTCA (Seq ID No 77)	NM_001277	Choline kinase (CHK)
JSZ45A	GCCCGCATGTCTACT TTTGTG (Seq ID No 20)	TGAAGTCAGGGACA GTCACCAA (Seq ID No 78)	NM_006230	DNA polymerase delta 2 regulatory subunit (POLD2)
JSZ44	TGTACGAGTCGGCCA AGTTG (Seq ID No 21)	GATTTGCAGGGCG ATGTCAT (Seq ID No 79)	NM_015675	Growth arrest and DNA-damage-inducible beta (GADD45B)
JSZ43A	AGGCTGAGCAAGCAG ATGGA (Seq ID No 22)	CTCACCAACCTGCA AAGTGCTA (Seq ID No 80)	NM_000824	Glycine receptor beta(GLRB)
JSZ42A	AAGGCTATGTTTACG TTTTACTCATTGT (Seq ID No 23)	TGAGCTGCCCCTCT GTCTCT (Seq ID No 81)	NM_022740	3' end of homeodomain interacting protein kinase 2 (HIPK2)
JSZ41	TGAGGCATCGCAATG TAAGACT (Seq ID No 24)	GGGCAGGGAGTTG AAGAAATT (Seq ID No 82)	NM_001276	Chitinase 3-like 1, cartilage glycoprotein-39 (CHI3L1)
JSZ40A	ACCTCCCCGCCGAGT TC (Seq ID No 25)	GAGGCTCCAGCTTA ACGGTATTT (Seq ID No 83)	NG_000006	Genomic alpha globin region (HBAalpha)
JSZ4	CCTCCGGGCGTGTGA A (Seq ID No 26)	CCTCTTGATTTCCC TTTGCTCTT (Seq ID No 84)	NM_139351	Bridging integrator 1(BIN1)
JSZ36	TCTTTGGCTTCAGAAT TGTTTTTAGA (Seq ID No 27)	CAGCAAACTCAACC CATCTCATT (Seq ID No 85)	NM_000794	Dopamine receptor D1(DRD1)
JSZ35	GCTATAATCCCCCTC AGGGCTAT (Seq ID No 28)	TGGAGGATTGATCT TGGCCATA (Seq ID No 86)	NM_004960	Fusion derived from t12;16 malignant liposarcoma (FUS)
JSZ34A	GTGAATCTGCACCAA GCATGA (Seq ID No 29)	CTAGTGAGAGGGTA GTCAGTAGCCACTT (Seq ID No 87)	NM_004083	DNA-damage-inducible transcript 3 (DDIT3)
JSZ33	GAGCCGGACTGGAC ATGGT (Seq ID No 30)	CCTGACAGGATCC GGAAGTCT (Seq ID No 88)	NM_000918	Pro collagen protein disulfide isomerase (P4HB)
JSZ32C	CAATGCCCTCTTTATT CTCTATTACACA (Seq ID No 31)	GTGGAAGGGCGGG AAGTC (Seq ID No 89)	NM_002309	Leukemia inhibitory factor (LIF)
JSZ31A	CCGAGTGTCCTCAGT ATATCAGGAA (Seq ID No 32)	CCATCTTTATCACC AGAATGAGGAA (Seq ID No 90)	NM_002964	S100 calcium binding protein A8 (S100A8)
JSZ30	TGCAGGCATGGTCCC TTAA (Seq ID No 33)	AGTCAGTTCATCTG GGCATCCT (Seq ID No 91)	NM_004428	Ephrin-A1 (EFNA1)
JSZ3	CAGCGACCTTCCTCA TCCA (Seq ID No 34)	AGCCTCTACTGCCA CCATCTTAA (Seq ID No 92)	NM_078467	Cyclin-dependent kinase inhibitor 1A p21/Cip1 (CDKN1A)
JSZ2A	GCAGGATGGACTCTT GCACAT (Seq ID No 35)	CAGCCAACAGTGTA GGTCTTGGT (Seq ID No 93)	NM_003254	Tissue inhibitor of metalloproteinase 1 (TIMP1)
JSZ29	TGAACTTCATCAGTTA AAGGCCAAT (Seq ID No 36)	CCCTTCGCCGGCTT CTT (Seq ID No 94)	NM_004746	Discs large homolog-associated protein 1 (DLGAP1)
JSZ28B	CCTCCGGGAAGTCTT GGAA (Seq ID No 37)	GGCCAAACGCACC GTTT (Seq ID No 95)	NM_000756	Corticotropin releasinghormone (CRH)
JSZ27B	GTGTTTGCCTCAGGC CAACT (Seq ID No 38)	CCAGTCCTATTGAA TGTGGGACTT (Seq ID No 96)	NM_016232	Interleukin 1receptor-like 1 (IL1RL1)
JSZ26	AGAGCCCTCCATCAC CTTCA (Seq ID No 39)	CAGCCCTATTCCAC TGAGTTAGTTT (Seq	AF209502	Calpain (CAPN3)

		ID No 97)		
JSZ25	AGCACAAGAGCCTCT CTCTGTCTAT (Seq ID No 40)	TGTACACGAACTCC TTGGCATT (Seq ID No 98)	NM_016272	Transducer of ERBB22(TOB2)
JSZ24	CTGGGTGAATGCCTT GAAGAA (Seq ID No 41)	ACTTTATGCTCCGA GGTGGTACA (Seq ID No 99)	NM_005393	Plexin B3 (PLXNB3)
JSZ23	GGTACCAGCCTTGGA TACTCCAT (Seq ID No 42)	TTCCGGGCTCAGCA TCAT (Seq ID No 100)	NM_004353	Serine or cysteine proteinase inhibitor clade H (SERPINH1)
JSZ22	CCTCGAAATGGACCC CAACT (Seq ID No 43)	GCAGCCCTGGGCA CACT (Seq ID No 101)	NM_005952	Metallothionein 1X(MT1X)
JSZ21	ACGTATCATGCACCA ACTGTGAA (Seq ID No 44)	TCTGGAACAGTCAT TTCCAGTGTT (Seq ID No 102)	XM_030707	KIAA0620 protein (KIAA0620)
JSZ20	AAGAAGAAGTGACCA AGGAGGAGTT (Seq ID No 45)	AGATGGGTTGTGAA GCAATGAGT (Seq ID No 103)	NM_006501	Myelin-associatedoligodendrocyte basic protein (MOBP)
JSZ81	AGAGGAGCGGCAGG AGTATGT(Seq ID No 46)	CTTGAAACTGCCCA AAATTCCA(Seq ID No 104)	NM_004613	Transglutaminase (TGM2)
JSZ18	CCTTCCTCTCTGCAA TGACCTT (Seq ID No 47)	GAGAACTCCTGGTG GACCCTAGT (Seq ID No 105)	NM_005567	Lectingalactoside-bindingsoluble3binding protein (LGALS3BP)
JSZ17	GCGCCCATTGATGAG CAT (Seq ID No.48)	CATCCTCCCACAGG CCTTT (Seq ID No 106)	NM_138924	GuanidinoacetateN-methyltransferase (GAMT)
JSZ16	ACCCCTGCCTTTGAT TGTCA (Seq ID No 49)	GAGAATAACTTAGA TCCGTGCAATAAAT AA (Seq ID No 107)	NM_012323	V-maf musculo aponeurotic fibrosarcoma oncogene homolog F (MAFF)
JSZ15	CCTGCTAAGAAGCTG ACTAATGCA (Seq ID No 50)	GAGTGGCTTCTCAG GCTGATCT (Seq ID No 108)	NM_032978	Dystro brevin alpha (DTNA)
JSZ14A	AAGCCTCAGCAGTTC TTTGGATT (Seq ID No 51)	TCATAATTCTGCATT GCACTCCTT (Seq ID No 109)	NM_003182	Tachykinin precursor 1:substance K and P,neurokinin 1and 2,neuromedin L, neurokinin alpha, K and gamma (TAC1)
JSZ13A	CCATCAAGACGGAGC TGACA (Seq ID No 52)	CCTTCTTCTTGCCA TCTGGATT (Seq ID No 110)	NM_007367	RNA binding protein (RALY)
JSZ12A	TAAGAATGGAGCAGT ACATGGGAAA (Seq ID No 53)	GGGACGCTGTGTC TCTCCAA (Seq ID No 111)	NM_000731	Cholecystokinin B receptor(CCKBR)
JSZ11	GTTCAGAGAGATAGG TGAGCTCTACCT (Seq ID No 54)	GGTGAAGGCTTCCT CAATGC (Seq ID No 112)	NM_001085	Serine or cysteine proteinase inhibitor clade A member 3 (SERPINA3)
JSZ10C	TCCTCAACACACCCA AGAAGCT (Seq ID No 55)	GAGAACGGCGGGT TCCA (Seq ID No 113)	NM_006185	Nuclear mitotic apparatus protein 1 (NUMA1)
JSZ1	GGAACTTTTCTATTAC AATCGCTTAGGA (Seq ID No 56)	CAGAGCGGGTGGG TCAGA (Seq ID No 114)	NM_006494	Ets2 repressor factor (ERF)
GAPDH SD	ATGGGGAAGGTGAAG GTCG (Seq ID No 57)	TAAAAGCAGCCCTG GTGACC (Seq ID No 115)	NM_002046	Glyceraldehyde-3-phosphate dehydrogenase (GAPD)
Actin2	AAGGATTCCTATGTG GGCGA (Seq ID No 58)	TCCATGTCGTCCCA GTTGGT (Seq ID No 116)	NM_001101	beta actin (ACTB)

RNA levels are then measured using Q-PCR. Briefly, cDNA is synthesized using random hexamers, diluted in a master mix containing TAQ polymerase, SybrGreen™ (Molecular Probes, Inc., Eugene, Oregon), unlabeled nucleotides, buffer and water. The mixture is aliquotted into TaqMan® plates (Perkin Elmer) and

pairs of oligonucleotides are added to the appropriate wells. Each sample is assayed in at least duplicate wells and every sample is assayed with every oligonucleotide pair where the transcriptase is omitted from the first reaction (noRT controls). The threshold cycle (C_T) is calculated using Perkin Elmer software ABI Prism ® 7700 Sequence Detection System Revison B. The C_T value is defined as the cycle at which a statistically significant increase in fluorescence (from the SybrGreen™) is detected. A lower C_T value is indicative of a higher mRNA concentration.

cDNA is separately prepared from a subset of 16 N (normal) and 16 S (schizophrenic) samples according to conventional methods. Yield is estimated using PicoGreen™ (Molecular Probes, Inc., Eugene, Oregon) assays. All the genes were measured by Q-PCR run on the individual cDNA samples. The individual C_T values for these genes relative to the actin level are examined and t-test and Kruskal Wallace p-values are calculated to test the null hypothesis that the two samples N and S are derived from the same population. Data indicate that thirteen genes are found to be differentially expressed between all the normal and schizophrenic anterior cingulate samples. These genes are listed in Table 3

Table 3

Genes upregulated in all schizophrenics relative to all the normals		
Oligopair	RefSeq or GenBank IDs	GeneName
SZ1-29	NM_003651	Cold shock domain protein A (CSDA)*
SZ1-25	NM_007021	Decidual protein induced by progesterone (DEPP)*
SZ1-11	NM_001124	Adrenomedullin (ADM)*
JSZ56B	NM_002982	Small inducible cytokineA2 (SCYA2)
JSZ44	NM_015675	Growth arrest and DNA-damage-inducible beta (GADD45B)
JSZ34A	NM_004083	DNA-damage-inducible transcript 3 (DDIT3)
JSZ31A	NM_002964	S100 calcium binding protein A8 (S100A8)
JSZ3	NM_078467	Cyclin-dependent kinase inhibitor 1A p21/Cip1 (CDKN1A)
JSZ27B	NM_016232	Interleukin 1receptor-like 1 (IL1RL1)
JSZ25	NM_016272	Transducer of ERBB2 (TOB2)
JSZ81	NM_004613	Transglutaminase (TGM2)
JSZ16	NM_012323	V-maf musculo aponeurotic fibrosarcoma oncogene homolog F (MAFF)

JSZ11	NM_001085	Serine or cysteine proteinase inhibitor clade A member 3 (SERPINA3)

*Interestingly, three genes that we identified previously as being associated with schizophrenia (decidual protein induced by progesterone (DEPP), adrenomedullin and cold shock domain protein A (cdsA)) are detected in these experiments, confirming the validity of the data disclosed herein.

Hierarchical clustering of the Q-PCR data showed that of the 16 S samples, seven formed a tight cluster. This indicates that based on expression levels of these 56 measured genes, these seven schizophrenic patients are more similar to one another than to any of the other patients or the normal controls. As such, the seven schizophrenic patients may define a subset of the disease, particularly since when these seven patients were compared to the rest of the other S and N patients, the above set of 13 genes as well as a further 9 genes, were significantly differentially regulated. These additional genes are listed in Table 4

Table 4

Genes upregulated in 7 schizophrenics		
Oligopair	RefSeq or GenBank IDs	GeneName
JSZ53C	NM_001511	GRO1 oncogene melanoma growth stimulating activity alpha (GRO1)
JSZ50	NM_000591	CD14 antigen (CD14)
JSZ49	NM_015319	Tensin 2 (KIAA1075)
JSZ41	NM_001276	Chitinase 3-like 1, cartilage glycoprotein-39 (CHI3L1)
JSZ23	NM_004353	Serine or cysteine proteinase inhibitor clade H (SERPINH1)
JSZ22	NM_005952	Metallothionein 1X(MT1X)
JSZ21	XM_030707	KIAA0620 protein (KIAA0620)
JSZ2A	NM_003254	Tissue inhibitor of metalloproteinase 1 (TIMP1)
JSZ10C	NM_006185	Nuclear mitotic apparatus protein 1 (NUMA1)

cDNA sequences

NM_002982: Small inducible cytokineA2 (SCYA2)
GGAACCGAGAGGCTGAGACTAACCCAGAAACATCCAATTCTCAAACTGAAGCTCGCACTCTCGC
CTCCAGCATGAAAGTCTCTGCCGCCCTTCTGTGCCTGCTGCTCATAGCAGCCACCTTCATTCCCC
AAGGGCTCGCTCAGCCAGATGCAATCAATGCCCCAGTCACCTGCTGTTATAACTTCACCAATAGG
AAGATCTCAGTGCAGAGGCTCGCGAGCTATAGAAGAATCACCAGCAGCAAGTGTCCCAAAGAAG
CTGTGATCTTCAAGACCATTGTGGCCAAGGAGATCTGTGCTGACCCCAAGCAGAAGTGGGTTCA
GGATTCCATGGACCACCTGGACAAGCAAACCCAAACTCCGAAGACTTGAACACTCACTCCACAAC
CCAAGAATCTGCAGCTAACTTATTTTCCCCTAGCTTTCCCCAGACACCCTGTTTTATTTTATTATAA
TGAATTTTGTTTGTTGATGTGAAACATTATGCCTTAAGTAATGTTAATTCTTATTTAAGTTATTGATG
TTTTAAGTTTATCTTTCATGGTACTAGTGTTTTTTAGATACAGAGACTTGGGGAAATTGCTTTTCCT
CTTGAACCACAGTTCTACCCCTGGGATGTTTTGAGGGTCTTTGCAAGAATCATTAATACAAAGAAT
TTTTTTTAACATTCCAATGCATTGCTAAAATATTATTGTGGAAATGAATATTTTGTAACTATTACACC
AAATAAATATATTTTTGTACAAAAAAAAAAAAAA Seq. ID No. 117

NM_015675: Growth arrest and DNA-damage-inducible beta (GADD45B)
CTAGCTCTGTGGGAAGGTTTTGGGCTCTCTGGCTCGGATTTTGCAATTTCTCCCTGGGGACTGCC
GTGGAGCCGCATCCACTGTGGATTATAATTGCAACATGACGCTGGAAGAGCTCGTGGCGTGCGA
CAACGCGGCGCAGAAGATGCAGACGGTGACCGCCGCGGTGGAGGAGCTTTTGGTGGCCGCTCA
GCGCCAGGATCGCCTCACAGTGGGGGTGTACGAGTCGGCCAAGTTGATGAATGTGGACCCAGA
CAGCGTGGTCCTCTGCCTCTTGGCCATTGACGAGGAGGAGGAGGATGACATCGCCCTGCAAATC
CACTTCACGCTCATCCAGTCCTTCTGCTGTGACAACGACATCAACATCGTGCGGGTGTCGGGCAA
TGCGCGCCTGGCGCAGCTCCTGGGAGAGCCGGCCGAGACCCAGGGCACCACCGAGGCCCGAG
ACCTCCACTGTCTTCCCTTCCTACAGAACCCTCACACGGACGCCTGGAAGAGCCACGGCTTGGT
GGAGGTGGCCAGCTACTGCGAAGAAAGCCGGGGCAACAACCAGTGGGTCCCCTACATCTCTCTT
CAGGAACGCTGAGGCCCTTCCCAGCAGCAGAATCTGTTGAGTTGCTGCCAACAAACAAAAAATAC
AATAAATATTTGAACCCCCTCCCCCCCAGCACAACCCCCCCAAAACAACCCAACCCACGAGGACC
ATCGGGGGCAGGTCGTTGGAGACTGAAGAGAAAGAGAGAGAGGAGAAGGGAGTGAGGGGCCG
CTGCCGCCTTCCCCATCACGGAGGGTCCAGACTGTCCACTCGGGGGTGGAGTGAGACTGACTG
CAAGCCCCACCCTCCTTGAGACTGGAGCTGAGCGTCTGCATACGAGAGACTTGGTTGAAACTTG
GTTGGTCCTTGTCTGCACCCTCGACAAGACCACACTTTGGGACTTGGGAGCTGGGGCTGAAGTT
GCTCTGTACCCATGAACTCCCAGTTTGCGAATTAATAAGAGACAATCTATTTTGTTACTTGCACTT
GTTATTCGAACCACTGAGAGCGAGATGGGAAGCATAGATATCTATATTTTTATTTCTACTATGAGG
GCCTTGTAATAAATTTCTAAAGCCTCAAAAAA Seq ID No. 118

NM_002964: S100 calcium binding protein A8 (S100A8)
ATGTCTCTTGTCAGCTGTCTTTCAGAAGACCTGGTGGGGCAAGTCCGTGGGCATCATGTTGACCG
AGCTGGAGAAAGCCTTGAACTCTATCATCGACGTCTACCACAAGTACTCCCTGATAAAGGGGAAT
TTCCATGCCGTCTACAGGGATGACCTGAAGAAATTGCTAGAGACCGAGTGTCCTCAGTATATCAG
GAAAAAGGGTGCAGACGTCTGGTTCAAAGAGTTGGATATCAACACTGATGGTGCAGTTAACTTCC
AGGAGTTCCTCATTCTGGTGATAAAGATGGGCGTGGCAGCCCACAAAAAAAGCCATGAAGAAAG
CCACAAAGAGTAGCTGAGTTACTGGGCCCAGAGGCTGGGCCCCTGGACATGTACCTGCAGAATA
ATAAAGTCATCAATACCTCAAAAAAAAAAAAAAAAAAAA Seq. ID No. 119

NM_078467: Cyclin-dependent kinase inhibitor 1A p21/Cip1 (CDKN1A)
AGCTGAGGTGTGAGCAGCTGCCGAAGTCAGTTCCTTGTGGAGCCGGAGCTGGGCGCGGATTCG
CCGAGGCACCGAGGCACTCAGAGGAGGTGAGAGAGCGGCGGCAGACAACAGGGGACCCCGGG
CCGGCGGCCCAGAGCCGAGCCAAGCGTGCCCGCGTGTGTCCCTGCGTGTCCGCGAGGATGCG
TGTTCGCGGGTGTGTGCTGCGTTCACAGGTGTTTCTGCGGCAGGCGCCATGTCAGAACCGGCTG
GGGATGTCCGTCAGAACCCATGCGGCAGCAAGGCCTGCCGCCGCCTCTTCGGCCCAGTGGACA
GCGAGCAGCTGAGCCGCGACTGTGATGCGCTAATGGCGGGCTGCATCCAGGAGGCCCGTGAGC

GATGGAACTTCGACTTTGTCACCGAGACACCACTGGAGGGTGACTTCGCCTGGGAGCGTGTGCG
GGGCCTTGGCCTGCCCAAGCTCTACCTTCCCACGGGGCCCCGGCGAGGCCGGGATGAGTTGGG
AGGAGGCAGGCGGCCTGGCACCTCACCTGCTCTGCTGCAGGGGACAGCAGAGGAAGACCATGT
GGACCTGTCACTGTCTTGTACCCTTGTGCCTCGCTCAGGGGAGCAGGCTGAAGGGTCCCCAGGT
GGACCTGGAGACTCTCAGGGTCGAAAACGGCGGCAGACCAGCATGACAGATTTCTACCACTCCA
AACGCCGGCTGATCTTCTCCAAGAGGAAGCCCTAATCCGCCCACAGGAAGCCTGCAGTCCTGGA
AGCGCGAGGGCCTCAAAGGCCCGCTCTACATCTTCTGCCTTAGTCTCAGTTTGTGTGTCTTAATT
ATTATTTGTGTTTTAATTTAAACACCTCCTCATGTACATACCCTGGCCGCCCCCTGCCCCCCAGCC
TCTGGCATTAGAATTATTTAAACAAAAACTAGGCGGTTGAATGAGAGGTTCCTAAGAGTGCTGGG
CATTTTTATTTTATGAAATACTATTTAAAGCCTCCTCATCCCGTGTTCTCCTTTTCCTCTCTCCCGG
AGGTTGGGTGGGCCGGCTTCATGCCAGCTACTTCCTCCTCCCCACTTGTCCGCTGGGTGGTACC
CTCTGGAGGGGTGTGGCTCCTTCCCATCGCTGTCACAGGCGGTTATGAAATTCACCCCCTTTCCT
GGACACTCAGACCTGAATTCTTTTTCATTTGAGAAGTAAACAGATGGCACTTTGAAGGGGCCTCA
CCGAGTGGGGGCATCATCAAAAACTTTGGAGTCCCCTCACCTCCTCTAAGGTTGGGCAGGGTGA
CCCTGAAGTGAGCACAGCCTAGGGCTGAGCTGGGGACCTGGTACCCTCCTGGCTCTTGATACCC
CCCTCTGTCTTGTGAAGGCAGGGGGAAGGTGGGGTCCTGGAGCAGACCACCCCGCCTGCCCTC
ATGGCCCCTCTGACCTGCACTGGGGAGCCCGTCTCAGTGTTGAGCCTTTTCCCTCTTTGGCTCC
CCTGTACCTTTTGAGGAGCCCCAGCTACCCTTCTTCTCCAGCTGGGCTCTGCAATTCCCCTCTGC
TGCTGTCCCTCCCCCTTGTCCTTTCCCTTCAGTACCCTCTCAGCTCCAGGTGGCTCTGAGGTGCC
TGTCCCACCCCCACCCCCAGCTCAATGGACTGGAAGGGGAAGGGACACACAAGAAGAAGGGCA
CCCTAGTTCTACCTCAGGCAGCTCAAGCAGCGACCGCCCCCTCCTCTAGCTGTGGGGGTGAGG
GTCCCATGTGGTGGCACAGGCCCCCTTGAGTGGGGTTATCTCTGTGTTAGGGGTATATGATGGG
GGAGTAGATCTTTCTAGGAGGGAGACACTGGCCCCTCAAATCGTCCAGCGACCTTCCTCATCCA
CCCCATCCCTCCCCAGTTCATTGCACTTTGATTAGCAGCGGAACAAGGAGTCAGACATTTTAAGA
TGGTGGCAGTAGAGGCTATGGACAGGGCATGCCACGTGGGCTCATATGGGGCTGGGAGTAGTT
GTCTTTCCTGGCACTAACGTTGAGCCCCTGGAGGCACTGAAGTGCTTAGTGTACTTGGAGTATTG
GGGTCTGACCCCAAACACCTTCCAGCTCCTGTAACATACTGGCCTGGACTGTTTTCTCTCGGCTC
CCCATGTGTCCTGGTTCCCGTTTCTCCACCTAGACTGTAAACCTCTCGAGGGCAGGGACCACAC
CCTGTACTGTTCTGTGTCTTTCACAGCTCCTCCCACAATGCTGAATATACAGCAGGTGCTCAATAA
ATGATTCTTAGTGACTTTAAAAAAAAAAAAAAAAAAA Seq. ID No.120

NM_016232: Interleukin 1receptor-like 1 (IL1RL1)
ATGGGGTTTTGGATCTTAGCAATTCTCACAATTCTCATGTATTCCACAGCAGCAAAGTTTAGTAAA
CAATCATGGGGCCTGGAAAATGAGGCTTTAATTGTAAGATGTCCTAGACAAGGAAAACCTAGTTA
CACCGTGGATTGGTATTACTCACAAACAAACAAAAGTATTCCCACTCAGGAAAGAAATCGTGTGTT
TGCCTCAGGCCAACTTCTGAAGTTTCTACCAGCTGAAGTTGCTGATTCTGGTATTTATACCTGTAT
TGTCAGAAGTCCCACATTCAATAGGACTGGATATGCGAATGTCACCATATATAAAAAACAATCAGA
TTGCAATGTTCCAGATTATTTGATGTATTCAACAGTATCTGGATCAGAAAAAAATTCCAAAATTTAT
TGTCCTACCATTGACCTCTACAACTGGACAGCACCTCTTGAGTGGTTTAAGAATTGTCAGGCTCTT
CAAGGATCAAGGTACAGGGCGCACAAGTCATTTTTGGTCATTGATAATGTGATGACTGAGGACGC
AGGTGATTACACCTGTAAATTTATACACAATGAAAATGGAGCCAATTATAGTGTGACGGCGACCA
GGTCCTTCACGGTCAAGGATGAGCAAGGCTTTTCTCTGTTTCCAGTAATCGGAGCCCCTGCACAA
AATGAAATAAAGGAAGTGGAAATTGGAAAAAACGCAAACCTAACTTGCTCTGCTTGTTTTGGAAAA
GGCACTCAGTTCTTGGCTGCCGTCCTGTGGCAGCTTAATGGAACAAAAATTACAGACTTTGGTGA
ACCAAGAATTCAACAAGAGGAAGGGCAAAATCAAAGTTTCAGCAATGGGCTGGCTTGTCTAGACA
TGGTTTTAAGAATAGCTGACGTGAAGGAAGAGGATTTATTGCTGCAGTACGACTGTCTGGCCCTG
AATTTGCATGGCTTGAGAAGGCACACCGTAAGACTAAGTAGGAAAAATCCAATTGATCATCATAG
CATCTACTGCATAATTGCAGTATGTAGTGTATTTTTAATGCTAATCAATGTCCTGGTTATCATCCTA
AAAATGTTCTGGATTGAGGCCACTCTGCTCTGGAGAGACATAGCTAAACCTTACAAGACTAGGAA
TGATGGAAAGCTCTATGATGCTTATGTTGTCTACCCACGGAACTACAAATCCAGTACAGATGGGG
CCAGTCGTGTAGAGCACTTTGTTCACCAGATTCTGCCTGATGTTCTTGAAAATAAATGTGGCTATA
CCTTATGCATTTATGGGAGAGATATGCTACCTGGAGAAGATGTAGTCACTGCAGTGGAAACCAAC
ATACGAAAGAGCAGGCGGCACATTTTCATCCTGACCCCTCAGATCACTCACAATAAGGAGTTTGC
CTACGAGCAGGAGGTTGCCCTGCACTGTGCCCTCATCCAGAACGACGCCAAGGTGATACTTATT
GAGATGGAGGCTCTGAGCGAGCTGGACATGCTGCAGGCTGAGGCGCTTCAGGACTCCCTC
CAGCATCTTATGAAAGTACAGGGGACCATCAAGTGGAGGGAGGACCACATTGCCAATAAAAGGT
CCCTGAATTCCAAATTCTGGAAGCACGTGAGGTACCAAATGCCTGTGCCAAGCAAAATTCCCAGA

AAGGCCTCTAGTTTGACTCCCTTGGCTGCCCAGAAGCAATAG Seq. ID No.121

NM_004613: Transglutaminase 2 (TGM2)
AACAGGCGTGACGCCAGTTCTAAACTTGAAACAAAACAAAACTTCAAAGTACACCAAAATAGAACCTCCT
TAAAGCATAAATCTCACGGAGGGTCTCGGCCGCCAGTGGAAGGAGCCACCGCCCCCGCCCCGACCATGGC
CGAGGAGCTGGTCTTAGAGAGGTGTGATCTGGAGCTGGAGACCAATGGCCGAGACCACCACACGGCCGAC
CTGTGCCGGGAGAAGCTGGTGGTGCGACGGGGCCAGCCCTTCTGGCTGACCCTGCACTTTGAGGGCCGCA
ACTACCAGGCCAGTGTAGACAGTCTCACCTTCAGTGTCGTGACCGGCCCAGCCCCTAGCCAGGAGGCCGG
GACCAAGGCCCGTTTTCCACTAAGAGATGCTGTGGAGGAGGGTGACTGGACAGCCACCGTGGTGGACCAG
CAAGACTGCACCCTCTCGCTGCAGCTCACCACCCCGGCCAACGCCCCCATCGGCCTGTATCGCCTCAGCC
TGGAGGCCTCCACTGGCTACCAGGGATCCAGCTTTGTGCTGGGCCACTTCATTTTGCTCTTCAACGCCTG
GTGCCCAGCGGATGCTGTGTACCTGGACTCGGAAGAGGAGCGGCAGGAGTATGTCCTCACCCAGCAGGGC
TTTATCTACCAGGGCTCGGCCAAGTTCATCAAGAACATACCTTGGAATTTTGGGCAGTTTCAAGATGGGA
TCCTAGACATCTGCCTGATCCTTCTAGATGTCAACCCCAAGTTCCTGAAGAACGCCGGCCGTGACTGCTC
CCGGCGCAGCAGCCCCGTCTACGTGGGCCGGGTGGGTAGTGGCATGGTCAACTGCAACGATGACCAGGGT
GTGCTGCTGGGACGCTGGGACAACAACTACGGGGACGGCGTCAGCCCCATGTCCTGGATCGGCAGCGTGG
ACATCCTGCGGCGCTGGAAGAACCACGGCTGCCAGCGCGTCAAGTATGGCCAGTGCTGGGTCTTCGCCGC
CGTGGCCTGCACAGTGCTGAGGTGCCTAGGCATCCCTACCCGCGTCGTGACCAACTACAACTCGGCCCAT
GACCAGAACAGCAACCTTCTCATCGAGTACTTCCGCAATGAGTTTGGGGAGATCCAGGGTGACAAGAGCG
AGATGATCTGGAACTTCCACTGCTGGGTGGAGTCGTGGATGACCAGGCCGGACCTGCAGCCGGGGTACGA
GGGCTGGCAGGCCCTGGACCCAACGCCCCAGGAGAAGAGCGAAGGAACGTACTGCTGTGGCCCAGTTCCA
GTTCGTGCCATCAAGGAGGGCGACCTGAGCACCAAGTACGATGCGCCCTTTGTCTTTGCGGAGGTCAATG
CCGACGTGGTAGACTGGATCCAGCAGGACGATGGGTCTGTGCACAAATCCATCAACCGTTCCCTGATCGT
TGGGCTGAAGATCAGCACTAAGAGCGTGGGCCGAGACGAGCGGGAGGATATCACCCACACCTACAAATAC
CCAGAGGGGTCCTCAGAGGAGAGGGAGGCCTTCACAAGGGCGAACCACCTGAACAAACTGGCCGAGAAGG
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TACAATGGGATCTTGGGGCCCGAGTGTGGCACCAAGTACCTGCTCAACCTAACCCTGGAGCCTTTCTCTG
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TCCGGAATGTCATCATTGGCCCCGCCTAAGGGACCCCTGCTCCCAGCCTGCTGAGAGCCCCCACCTTGAT
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GCAGCCTATGGGGGCTCTCGGAAATGGAATGTGCCCCTGGCCCATCTCAGCCTCCTGAGCCTGTGGGTCC
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ACCCACCATATTGTTTGATCTACTTCATAGCTCCTTGGAGCAGGCAAAAAAGGGACAGCATGCCCTTGGC
TGGATCAGGAATCCAGCTCCCTAGACTGCATCCCGTACCTCTTCCCATGACTGCACCCAGCTCCAGGGGC
CCTTGGGACACCCAGAGCTGGGTGGGGACAGTGATAGGCCCAAGGTCCCCTCCACATCCCAGCAGCCCAA
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GCTGGGGCTCACGGGGCCTCCAACCCATTTAATCACCATGGGAAACTGTTGTGGGCGCTGCTTCCAGGAT
AAGGAGACTGAGGCTTAGAGAGAGGAGGCAGCCCCCTCCACACCAGTGGCCTCGTGGTTATAAGCAAGGC
TGGGTAATGTGAAGGCCCAAGAGCAGAGTCTGGGCCTCTGACTCTGAGTCCACTGCTCCATTTATAACCC
CAGCCTGACCTGAGACTGTCGCAGAGGCTGTCTGGGGCCTTTATCAAAAAAAGACTCAGCCAAGACAAGG
AGGTAGAGAGGGGACTGGGGGACTGGGAGTCAGAGCCCTGGCTGGGTTCAGGTCCCACGTCTGGCCAGCG
ACTGCCTTCTCCTCTCTGGGCCTTTGTTTCCTTGTTGGTCAGAGGAGTGATTGAACCTGCTCATCTCCAA
GGATCCTCTCCACTCCATGTTTGCAATACACAATTCC

Seq ID No 122

NM_012323: V-maf musculo aponeurotic fibrosarcoma oncogene homolog F (MAFF)
AGTAATTCCGGGAAGCTCGCCTTACAACTCCGCGCGGCCTCGGCCCCCTGCGCCGCCCGCCCC

ACAACAAAACTCAGCGCAGCGCTCCCGGGCGCCCGGTTCAGAGCGACCTGCGGCTCAGAGCGG
AGGGGAGACTGACCGGAGCGCGGATCGGGACAGCGGCCGGGACAGCGGCGAGACGCGCGTGT
GTGAGCGCGCCGGACCAAGCGGGCCCAGAAGCGGGTCTGCAGCCCAGAGGGC
ACCTTCTGCAAACATGTCTGTGGATCCCCTATCCAGCAAAGCTCTAAAGATCAAGCGAGAGCTGA
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CGGCATCTGCGCGGGCTCTCCGCCGAGGAGGTGACACGGCTCAAGCAGCGGCGCCGCACACT
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CACCCCGGGCTCGGGGTCTGGCCCCGCCCACGGCCCGGACCCCGCCCACGGCCCGGCCTCCT
GCTCCTAGTGCCCGCCCCCGCCATGCCTCAGCCACGCCCCTCCGGCCTCAGCTCCCTCCCCAA
AGTGCCTGAGCGCCGCCTCTGTGCCCAGGTCCCATTTCTCTGCAGCACTGGCCCCTTGGTGCAC
ACACATTCCCTTCGTGGGCCCTGTCTTCCTCTTGCAGCCCCCCAAACTGGGACCGAATGACCCT
GGGAAGGGGAACTTGGGTAGGTTGGGGATGGGGCAGAGGTCTGGATCTGGGATCGCCCTTGGC
TGAAAGTTTAGCCTTTTTAGATTGAGAGATACAGAGCCGGCTTAGAGAACAGCTGTTGGGGGAGA
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GATGCCCCCACCTCTGGTTGGGCCTGGGGGTGCCTGGCCTTCCGAAACTAAAAGAGTGGGTGG
GAAGACTAGTGAAACCCAGTTCACGGATGGGGAAACAGGCCTGAGGTCACATTTCACTTAGTGG
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TGCAAGGGAGGGGGAGAGAAGCAGAGGGAGAGAGAAGGTGACACGGATGGAAGAGTGGGAAG
GAGCTGGCCTGGCTCAGCCCTAGGCTGTCCCTGCAGCCAGGGTGTCCGGGGGCTGGCCAGTCA
GAGAAAGGGGGCCATGGACTGCTGTGGCAAATAGGGAGACAAGGAGACAGACCCTGCAGTCCT
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CCCCAACACTGTCCACACTGCCCCTCCCCACTGTTTATTTATTGCACGGATCTAAGTTATTCTCCC
CAGCCAGAGCCCGAGCTCCTGCTCCCTGGGAAAAGTGGCGTATGGCCCTGAGCTGGGCTTTATA
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AATACAAAAATTAGCCATGCATGGTGGCTCATGCCTGTAGTCCCAGCTACTTGGGAGGCTGAGGC
AGGAGGATCACTTAAGCCCAGAAGGCAGAGGTTGTAGTGAGCTGAGATCGCACCACTGCACTCC
AGCCTGGGCAACATAGCAAAATCCTGTCTCAAAAAAAAAGTTAAAAAATATTGCCCGGCTCCTAGA
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AGTCAAATTCTTTCTTTTTCCACAGAGAAAAAAA Seq. ID No.123

NM_001085: Serine or cysteine proteinase inhibitor clade A member 3 (SERPINA3)
GGAATTCCCTGGAGCAGAGTTGAGAATGGAGAGAATGTTACCTCTCCTGGCTCTGGGGCTCTTG
GCGGCTGGGTTCTGCCCTGCTGTCCTCTGCCACCCTAACAGCCCACTTGACGAGGAGAATCTGA
CCCAGGAGAACCAAGACCGAGGGACACACGTGGACCTCGGATTAGCCTCCGCCAACGTGGACT
TCGCTTTCAGCCTGTACAAGCAGTTAGTCCTGAAGGCCCTTGATAAGAATGTCATCTTCTCCCCA
CTGAGCATCTCCACCGCCTTGGCCTTCCTGTCTCTGGGGGCCCATAATACCACCCTGACAGAGA
TTCTCAAGGCCTCGAGTTCACCTCACGGAGACTTACTGAGGCAGAAATTCACTCAGAGCTTCCAG
CACCTCCGCGCACCCTCAATCAGTTCCAGCGATGAGCTGCAGCTGAGTATGGGAAATGCCATGT
TTGTCAAAGAGCAACTCAGTCTGCTGGACAGGTTCACGGAGGATGCCAAGAGGCTGTATGGCTC
CGAGGCCTTTGCCACTGACTTTCAGGACTCAGCTGCAGCTAAGAAGCTCATCAACGACTACGTGA
AGAATGGAACTAGGGGGAAAATCACAGATCTGATCAAGGACCCCGACTCGCAGACAATGATGGT
CCTGGTGAATTACATCTTCTTTAAAGCCAAATGGGAGATGCCCTTTGACCCCCAAGATACTCATCA
GTCAAGGTTCTACTTGAGCAAGAAAAAGTGGGTAATGGTGCCCATGATGAGTTTGCATCACCTGA
CTATACCTTACTTCCGGGACGAGGAGCTGTCCTGCACCGTGGTGGAGCTGAAGTACACAGGCAA
TGCCAGCGCACTCTTCATCCTCCCTGATCAAGACAAGATGGAGGAAGTGGAAGCCATGCTGCTC
CCAGAGACCCTGAAGCGGTGGAGAGACTCTCTGGAGTTCAGAGAGATAGGTGAGCTCTACCTGC
CAAAGTTTTCCATCTCGAGGGACTATAACCTGAACGACATACTTCTCCAGCTGGGCATTGAGGAA
GCCTTCACCAGCAAGGCTGACCTGTCAGGGATCACAGGGGCCAGGAACCTAGCAGTCTCCCAG
GTGGTCCATAAGGTCGTGTCTGATGTATTTGAGGAGGGCACAGAAGCATCTGCTGCCACAGCAG

TCAAAATCACCCTCCTTTCTGCATTAGTGGAGACAAGGACCATTGTGCGTTTCAACAGGCCCTTC
CTGATGATCATTGTCCCTACAGACACCCAGAACATCTTCTTCATGAGCAAAGTCACCAATCCCAG
CAAGCCTAGAGCTTGCATCAAGCAGTGGGGCTCTCAGTAAGGAACTTGGAATGCAAGCTGGATG
CCTGGGTCTCTGGGCACAGCTGGCCCCTGTGCACCGTAGTGGCCATGGCATGTGTGGCCCTGT
CTGCTTATCCTTGGAAGGTGACAGCGATTCCCTGTGAAGCTCTCACACGCACAGGGGCCCATGG
ACTCTTCAGTCTGGAGGGTCCTGGCCTCCTGACAGCAATAAATAATTTCGTTGGCC Seq. ID No.124

NM_001511: GRO1 oncogene melanoma growth stimulating activity alpha (GRO1)
CACAGAGCCCGGGCCGCAGGCACCTCCTCGCCAGCTCTTCCGCTCCTCTCACAGCCGCCAGAC
CCGCCTGCTGAGCCCCATGGCCCGCGCTGCTCTCTCCGCCGCCCCCAGCAATCCCCGGCTCCT
GCGAGTGGCACTGCTGCTCCTGCTCCTGGTAGCCGCTGGCCGGCGCGCAGCAGGAGCGTCCGT
GGCCACTGAACTGCGCTGCCAGTGCTTGCAGACCCTGCAGGGAATTCACCCCAAGAACATCCAA
AGTGTGAACGTGAAGTCCCCCGGACCCCACTGCGCCCAAACCGAAGTCATAGCCACACTCAAGA
ATGGGCGGAAAGCTTGCCTCAATCCTGCATCCCCCATAGTTAAGAAAATCATCGAAAAGATGCTG
AACAGTGACAAATCCAACTGACCAGAAGGGAGGAGGAAGCTCACTGGTGGCTGTTCCTGAAGGA
GGCCCTGCCCTTATAGGAACAGAAGAGGAAAGAGAGACACAGCTGCAGAGGCCACCTGGATTGT
GCCTAATGTGTTTGAGCATCGCTTAGGAGAAGTCTTCTATTTATTTATTTATTCATTAGTTTTGAAG
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ATTATATTCATTCTTTTTGAAATGTCAACCCCAAGTTAGTTCAATCTGGATTCATATTTAATTTGAAG
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CCACTGTGATAGAGGCTGGCGGATCCAAGCAAATGGCCAATGAGATCATTGTGAAGGCAGGGGA
ATGTATGTGCACATCTGTTTTGTAACTGTTTAGATGAATGTCAGTTGTTATTTATTGAAATGATTTC
ACAGTGTGTGGTCAACATTTCTCATGTTGAAACTTTAAGAACTAAAATGTTCTAAATATCCCTTGGA
CATTTTATGTCTTTCTTGTAAGGCATACTGCCTTGTTTAATGGTAGTTTTACAGTGTTTCTGGCTTA
GAACAAAGGGGCTTAATTATTGATGTTTTCATAGAGAATATAAAAATAAAGCACTTATAG Seq. ID No.125

NM_000591: CD14 antigen (CD14)
CCGGCCGGCCGAAGAGTTCACAAGTGTGAAGCCTGAAGCCGCCGGGTGCCGCTGTGTAGAAAG
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CGCGTCCTGCTTGTTGCTGCTGCTGCTGCCGCTGGTGCACGTCTCTGCGACCACGCCAGAACCT
TGTGAGCTGGACGATGAAGATTTCCGCTGCGTCTGCAACTTCTCCGAACCTCAGCCCGACTGGT
CCGAAGCCTTCCAGTGTGTGTCTGCAGTAGAGGTGGAGATCCATGCCGGCGGTCTCAACCTAGA
GCCGTTTCTAAAGCGCGTCGATGCGGACGCCGACCCGCGGCAGTATGCTGACACGGTCAAGGC
TCTCCGCGTGCGGCGGCTCACAGTGGGAGCCGCACAGGTTCCTGCTCAGCTACTGGTAGGCGC
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ACCATGCCTCCGCTGCCTCTGGAAGCCACAGGACTTGCACTTTCCAGCTTGCGCCTACGCAACG
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CTCTGTCCCCACAAGTTCCCGGCCATCCAGAATCTAGCGCTGCGCAACACAGGAATGGAGACGC
CCACAGGCGTGTGCGCCGCACTGGCGGCGGCAGGTGTGCAGCCCCACAGCCTAGACCTCAGCC
ACAACTCGCTGCGCGCCACCGTAAACCCTAGCGCTCCGAGATGCATGTGGTCCAGCGCCCTGAA
CTCCCTCAATCTGTCGTTCGCTGGGCTGGAACAGGTGCCTAAAGGACTGCCAGCCAAGCTCAGA
GTGCTCGATCTCAGCTGCAACAGACTGAACAGGGCGCCGCAGCCTGACGAGCTGCCCGAGGTG
GATAACCTGACACTGGACGGGAATCCCTTCCTGGTCCCTGGAACTGCCCTCCCCCACGAGGGCT
CAATGAACTCCGGCGTGGTCCCAGCCTGTGCACGTTCGACCCTGTCGGTGGGGGTGTCGGGAA
CCCTGGTGCTGCTCCAAGGGGCCCGGGGCTTTGCCTAAGATCCAAGACAGAATAATGAATGGAC
TCAAACTGCCTTGGCTTCAGGGGAGTCCCGTCAGGACGTTGAGGACTTTTCGACCAATTCAACCC
TTTGCCCCACCTTTATTAAAATCTTAAAC Seq. ID No.126

NM_015319: Tensin 2 (KIAA1075)
GCACATTCTTTCAAGTGACAGCTATAGCCTGTCCCAGGGGCTGCTGTCCACAGCTTGGGGCTGA
AGACTCCCAGGCCATTAACCCCTTAGCTTTTAGGAAGATTACTCCCCCTTTTTCAAGGCCCCATCC
ACCTCCCTCCCTTGACTCCCAGGACGGGAAGTTGGCCATGTTCCCAGGAGGGAGGCCGGAGGC

CCATGGATGGGGGTGGAGTATGTGTTGGGAGGGGGGACCTCCTGTCCAGTCCTCAGGCCCTGG
GACAGCTGCTGAGGAAGGAGAGCAGACCCAGGAGAGCCATGAAGCCTAGGAAAGCTGAGCCTC
ATAGCTTCCGGGAGAAGGTTTTCCGGAAGAAACCTCCAGTCTGTGCAGTATGTAAGGTGACCATC
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GTGACTTCAGCCTGTCAGGCCTTGCCTCCCGTGGAGTTGCGGCGAAACACGGCCCCAGTCAGG
CGCATAGAGCACCTGGGATCCACCAAATCTCTGAACCACTCAAAGCAGCGCAGCACTCTGCCCA
GGAGCTTCAGCCTGGACCCGCTCATGGAGCGGCGCTGGGACTTAGACCTCACCTACGTGACGG
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CCACAGCACGTGGTCGTACTATACTGCAAGGGAAACAAGGGCAAGCTTGGGGTCATCGTTTCTG
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CATCCCCATGCTGCCAGCCTTTGAACCTGGCACAGGCTTCCAGCCCTTCCTTAAAATCTACCAGT
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GCCGGGGCACAGACCGGACCCTCGTGTTCCGAGTCCAGTTCCACACCTGCACCATCCACGGAC
CACAGCTCACTTTCCCCAAGGACCAGCTTGACGAGGCCTGGACTGATGAGAGGTTCCCCTTCCA
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TGTCAGAGGGGCTATACCCCTACCCACCTGAGATGGGGAAACCAGCCACTGGGGACTTTGGCTA
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ATGCCCAGCCTGCGAGGAGAAGCTGGCGCTGCCTACAGCAGCCTTGTATGGACTGCGGCTGGA
GAGGGAGGCTGGAGAAGGGTGGGCAAGTGAGGCTGGCAAGCCTCTCCTGCACCCAGTGCGGC
CTGGGCACCCGCTGCCTCTGCTCTTGCCTGCCTGTGGGCATCACCATGCCCCGATGCCTGACTA
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CCCCGAAGGCAGGTATGGGCATCCAGGGTACCCTGCCCTGGTGACATACAGCTATGGAGGAGC
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AGAGGGTATCCCAGCCCTGGTGCCCACTCCCCACGGGCTGGCTCCATTTCCCCGGGCAGCCCG
CCCTATCCACAATCTAGGAAGCTGAGCTACGAGATCCCTACGGAGGAGGGAGGGGACAGGTACC
CATTGCCTGGGCACCTGGCCTCAGCAGGACCTTTGGCATCTGCAGAGTCGCTGGAGCCGGTGTC
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CCACCACACTTCCTGGCCTCCGCCACGCCCCCTGGCAAGGCCCTCGAGGCCCCCCCCGACAGCC
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AGATGACCTTGCATGTGAGCAGATGGCAGAGATGGGTGTGTGAGGGGTGAGGAGGCATCAGCA
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AGCCCCTGCCAGTTCCACCCAGCTGCAGGTGCCAGCACGGCAGGGATGGGAGAGGGGTGGGG
AGCGAGTCACTGCCTCCTCTGAGCAGAGATTCAGAGTAGGATCACATGAATAGGGGAAAAAAGA
GAGTCTATTTTTGTCTAATAATAAAGAATTTCTATAAACTTT Seq. ID No. 127

NM_001276: Chitinase 3-like 1, cartilage glycoprotein-39 (CHI3L1)
AGTGGAGTGGGACAGGTATATAAAGGAAGTACAGGGCCTGGGGAAGAGGCCCTGTCTAGGTAG
CTGGCACCAGGAGCCGTGGGCAAGGGAAGAGGCCACACCCTGCCCTGCTCTGCTGCAGCCAGA
ATGGGTGTGAAGGCGTCTCAAACAGGCTTTGTGGTCCTGGTGCTGCTCCAGTGCTGCTCTGCAT
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CATCGACACCTGGGAGTGGAATGATGTGACGCTCTACGGCATGCTCAACACACTCAAGAACAGG
AACCCCAACCTGAAGACTCTCTTGTCTGTCGGAGGATGGAACTTTGGGTCTCAAAGATTTTCCAA
GATAGCCTCCAACACCCAGAGTCGCCGGACTTTCATCAAGTCAGTACCGCCATTCCTGCGCACC
CATGGCTTTGATGGGCTGGACCTTGCCTGGCTCTACCCTGGACGGAGAGACAAACAGCATTTTA
CCACCCTAATCAAGGAAATGAAGGCCGAATTTATAAAGGAAGCCCAGCCAGGGAAAAAGCAGCT
CCTGCTCAGCGCAGCACTGTCTGCGGGGAAGGTCACCATTGACAGCAGCTATGACATTGCCAAG
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CAACCAGTGGGTAGGATACGACGACCAGGAAAGCGTCAAAAGCAAGGTGCAGTACCTGAAGGAT
AGGCAGCTGGCAGGCGCCATGGTATGGGCCCTGGACCTGGATGACTTCCAGGGCTCCTTCTGC
GGCCAGGATCTGCGCTTCCCTCTCACCAATGCCATCAAGGAT
GCACTCGCTGCAACGTAGCCCTCTGTTCTGCACACAGCACGGGGGCCAAGGATGCCCCGTCCC
CCTCTGGCTCCAGCTGGCCGGGAGCCTGATCACCTGCCCTGCTGAGTCCCAGGCTGAGCCTCA
GTCTCCCTCCCTTGGGGCCTATGCAGAGGTCCACAACACACAGATTTGAGCTCAGCCCTGGTGG
GCAGAGAGGTAGGGATGGGGCTGTGGGGATAGTGAGGCATCGCAATGTAAGACTCGGGATTAG
TACACACTTGTTGATGATTAATGGAAATGTTTACAGATCCCCAAGCCTGGCAAGGGAATTTCTTCA
ACTCCCTGCCCCCTAGCCCTCCTTATCAAAGGACACCATTTTGGCAAGCTCTATCACCAAGGAGC
CAAACATCCTACAAGACACAGTGACCATACTAATTATACCCCCTGCAAAGCCAGCTTGAAACCTTC
ACTTAGGAACGTAATCGTGTCCCCTATCCTACTTCCCCTTCCTAATTCCACAGCTGCTCAATAAAG
TACAAGAGTTTAACAGTGTGTTGGCGCTTTGCTTTGGTCTATCTTTGAGCGCCCACTAGACCCACT
GGACTCACCTCCCCCATCTCTTCTGGGTTCCTTCCTCTGAGCCTTGGGACCCCTGAGCTTGCAGA
GATGAAGGCCGCCATGTT Seq. ID No. 128

NM_004353: Serine or cysteine proteinase inhibitor clade H (SERPINH1)
GGTCCTCTGTGGTGCACAGCCCACCCCCCAGCCATGCGCTCTCTCCTTCTGGGCACCTTATGCC
TCCTGGCTGTGGCCCTGGCAGCCGAGGTGAAGAAACCTGTAGAGGCCGCAGCCCCTGGTACTG
CGGAGAAGCTGAGTTCCAAGGCGACCACACTGGCAGAGCCCAGCACAGGCCTGGCCTTCAGCC

TGTATCAGGCAATGGCCAAGGACCAGGCAGTGGAGAACATCCTGGTGTCACCCGTGGTGGTGG
CCTCGTCGCTGGGTCTCGTGTCGCTGGGCGGCAAGGCGACCACGGCGTCGCAGGCCAAGGCA
GTGCTGAGCGCCGAGCAGCTGCGCGACGAGGAGGTGCACGCCGGCCTGGGTGAGCTGCTGCG
CTCACTCAGCAACTCGACGGCGCGCAACGTGACCTGGAAGCTGGGCAGCCGACTGTACGGACC
CAGCTCAGTGAGCTTCGCTGATGACTTCGTGCGCAGCAGCAAGCAGCACTACAACTGCGAGCAC
TCCAAGATCAACTTCCCGGACAAGCGCAGCGCGCTGCAGTCCATCAACGAGTGGGCCGCGCAG
ACCACCGACGGCAAGCTGCCCGAGGTCACCAAGGACGTGGAGCGCACGGACGGCGCCCTGCTA
GTCAACGCCATGTTCTTCAAGCCACACTGGGATGAGAAATTCCACCACAAGATGGTGGACAACCG
TGGCTTCATGGTGACTCGGTCCTATACTGTGGGTGTTACGATGATGCACCGGACAGGCCTCTACA
ACTACTACGACGACGAGAAGGAGAAGCTGCAGCTGGTGGAGATGCCCCTGGCTCACAAGCTCTC
CAGCCTCATCATCCTCATGCCCCATCACGTGGAGCCTCTCGAGCGCCTTGAAAAGCTGCTAACCA
AAGAGCAGCTGAAGATCTGGATGGGGAAGATGCAGAAGAAGGCTGTTGCCATCTCCTTGCCCAA
GGGTGTGGTGGAGGTGACCCATGACCTGCAGAAACACCTGGCTGGGCTGGGCCTGACTGAGGC
CATTGACAAGAACAAGGCCGACTTATCACGCATGTCTGGCAAGAAGGATCTGTACCTGGCCAGT
GTGTTCCACGCCACCGCCTTTGAGTTGGACACAGATGGCAACCCCTTTGACCAGGACATCTACG
GGCGCGAGGAGCTGCGCAGCCCCAAGCTGTTCTACGCCGACCACCCCTTCATCTTCCTGGTGC
GGGACACCCAAAGCGGCTCCCTGCTATTCATTGGGCGCCTGGTCCGGCTCAAGGGTGACAAGAT
GCGAGACGAGTTATAGGGCCTCAGGGTGCACACAGGATGGCAGGAGGCATCCAAAGGCTCCTG
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TTCGAGCTCCACTTGGACATGGGCCCCAGATACCATGATGCTGAGCCCGGAAACTCCACATCCT
GTGGGACCTGGGCCATAGTCATTCTGCCTGCCCTGAAAGTCCCAGATCAAGCCTGCCTCAATCA
GTATTCATATTTATAGCCAGGTACCTTCTCACCTGTGAGACCAAATTGAGCTCGGGGGGGTCAGCC
AGCCCTCTTCTGACACTAAAACACCTCAGCTGCCTCCCCAGCTCTATCCCAACCTCTCCCAACTA
TAAAACTAGGTGCTGCAGCCTGGGACCAGGCACCCCCAGAATGACCTGGCCGCAGTGAGGCGA
TTGAGAAGGAGCTCCCAGGAGGGGCTTCTGGGAAGACCCTGGTCAAGAAGCATCGTCTGGCGTT
GTGGGGATGAACTTTTTGTTTTGTTTCTTCCTTTTTTAGTTCTTCAAGGAATGGGGGGCCAGGGG
GGCAATGAGCCTTTGTTGCTAATCAAATCCGGGACTTGTTTGTACGTTTTTTTTTCTCACTGAAAC
CTTTTCCAGTGCCAAAAAAAAA Seq. ID No.129

NM_005952: Metallothionein 1X(MT1X)
ACCACGCTTTTCATCTGTCCCGCTGCGTGTTTTCCTCTTGATCGGGAACTCCTGCTTCTCCTTGCC
TCGAAATGGACCCCAACTGCTCCTGCTCGCCTGTTGGCTCCTGTGCCTGTGCCGGCTCCTGCAA
ATGCAAAGAGTGCAAATGCACCTCCTGCAAGAAGAGCTGCTGCTCCTGCTGCCCTGTCGGCTGT
GCCAAGTGTGCCCAGGGCTGCATCTGCAAAGGGACGTCAGACAAGTGCAGCTGCTGTGCCTGA
Seq. ID No. 130

XM_030707: KIAA0620 protein (KIAA0620)
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GCCGCTGCTGTTGCTGCTGCTCCTGGGGGCGGCGCGGGCCGGCGCCCTGGAGATCCAGCGTC
GGTTCCCCTCGCCCACGCCCACCAACAACTTCGCCCTGGACGGCGCGGCGGGGACCGTGTACC
TGGCGGCCGTCAACCGCCTCTATCAGCTGTCGGGCGCCAACCTGAGCCTGGAGGCCGAGGCGG
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GAGCACCCGCGGCGCCTCACGGACAACTACAACAAGATCCTGCAGCTGGACCCCGGCCAGGGC
CTGGTAGTCGTGTGCGGGTCCATCTACCAGGGCTTCTGCCAGCTGCGGCGCCGGGGCAACATC
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CAGCATGCTGAACGTGGCGGCCAACCACCCGAACGCGTCCACCGTGGGGCTAGTTCTGCCTCC
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CCTTCTTCCCGCGCAACCGCAGCCTGGAGGACCACCGCTTCGAGAACACGCCCGAGATCGCCAT
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CTTGCAGCAGGACTGCACCAATTCCAGCCAGCAGCATTTCTGGACCAGTGCCAGCGAGGGCCCC
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GAACAACATCCGCACTGTGGCTCGGGTCCCAGGCCCTGCCTTTGGTCACCAGATTGCCTACTGC
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GCGATTGAGCCCCTGAGTGGCCCGTTGGACGGTGGGACCCTGCTGACCATCCGAGGAAGGAAC
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CCGGGGCCCTGAGCAACGCATCAGCGCCAGTGGACTTCTTCATCAATGGGCGGGCCTACGCAG
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CGCCTGGACTACCTCCCCAACCCACAGTTCTCTACGGCCAAGAGGGAGAAGTGGATCAAGCACC
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CGGCCCAACATGGAAGAGGGAATTAGCTTGTTCTCCTCACTACTCAACAACAAGCACTTCCTCAT
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TAACACGCTGGCCCATTACAAGATCCCTGAAGGTGCCTCCCTGGCCATGAGTCTCATAGACAAGA
AGGACAACACACTGGGCCGAGTGAAAGACTTGGACACAGAGAAGTATTTCCATTTGGTGCTGCCT
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CAAGGCCATTCTGAGTATCCGTGAAGACAAGCCCCCACTGGCTGTCAAGTACTTTTTCGACTTCC
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CAAGCACAAACTGGGGGGAGAGGAGGCCGCCAGCCCGGAGGCTGCAGCCCAGAAACTCTACCTC
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AATGTTATCTCCACTTCCCCCTCACCCGTAGAGGATCCTGGCCACAGACAGTTTCAAGTAGTGTC
AGATTTTTGTTGCTTGGGCGGCTGTTGGTAGAGTGGGCAGTGCCCGCGCCATGGGGTGCTCTGT
GGGCTTCTCCAGGAGCAGGGAGGGTGGAGGGGAGGGATGGGGGGCACAGGAGCTGGGAGCC
CCGTCTCCAGGAAAAGGAGAGGGGTTAAGATGCACCGAGGCTGTAGCTGGGCTACTTGATCTTG
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GCACCAACTGTGAATAGCTGCCGCTTGCGCAGAGGACCCGGGGAGGGGTCCCGAGAGGCTCCC
CATGCAACACTGGAAATGACTGTTCCAGAGAGCGGGCAGACCTGGCAGAGCGCCCCTGGCGCC
TGAGACTACCACCCACTCCGTTCCTGCCAGAAACGACCCTCTGTGGCCGATGGGCCATGCGGGC
CCCTCGCAGCCAACTCAGCCAGTGTTGGGACTGGCTCAGAGCCCATGGGGGCTGGAGGGGGGC
AGCTGGGACTCTGGAATCTTCTTTATAATAAAAGCCTTACGG
AC Seq. ID No.131

NM_003254: Tissue inhibitor of metalloproteinase 1 (TIMP1)
AGGGGCCTTAGCGTGCCGCATCGCCGAGATCCAGCGCCCAGAGAGACACCAGAGAACCCACCA
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GGCCTGCACCTGTGTCCCACCCCACCCACAGACGGCCTTCTGCAATTCCGACCTCGTCATCAGG
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GACCAAGATGTATAAAGGGTTCCAAGCCTTAGGGGATGCCGCTGACATCCGGTTCGTCTACACC
CCCGCCATGGAGAGTGTCTGCGGATACTTCCACAGGTCCCACAACCGCAGCGAGGAGTTTCTCA
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AGCCTGAGCTTAGCTCAGCGCCGGGGCTTCACCAAGACCTACACTGTTGGCTGTGAGGAATGCA
CAGTGTTTCCCTGTTTATCCATCCCCTGCAAACTGCAGAGTGGCACTCATTGCTTGTGGACGGAC
CAGCTCCTCCAAGGCTCTGAAAAGGGCTTCCAGTCCCGTCACCTTGCCTGCCTGCCTCGGGAGC
CAGGGCTGTGCACCTGGCAGTCCCTGCGGTCCCAGATAGCCTGAATCCTGCCCGGAGTGGAAC
TGAAGCCTGCACAGTGTCCACCCTGTTCCCACTCCCATCTTTCTTCCGGACAATGAAATAAAGAG
TTACCACCCAGC Seq. ID No.132

NM_006185: Nuclear mitotic apparatus protein 1 (NUMA1)
GCCCACGAAGAGGTACGATTCCGGAGAATCGCGAGGCAGAGCGGGAGCGCGCAGCCAGGTGG
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TGTCATTCTTAAATTTGTGCTGGACCATGAGGACGGGCTAAACCTTAATGAGGACCTAGAGAACT
TCCTACAGAAAGCTCCTGTGCCTTCTACCTGTTCTAGCACATTCCCTGAAGAGCTCTCCCCACCT
AGCCACCAGGCCAAGAGGGAGATTCGCTTCCTAGAGCTACAGAAGGTTGCCTCCTCTTCCAGTG
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GACTGGAACCGCATTGCAGAGCTGCAGCAGCGCAATCGAGTGTGCCCCCCACATCTGAAGACCT
GCTATCCCCTGGAGTCCAGGCCTTCCCTGAGCCTGGGCACCATCACAGATGAGGAGATGAAAAC
TGGAGACCCCCAAGAGACCCTGCGCCGAGCCAGCATGCAGCCAATCCAGATAGCCGAGGGCAC
TGGCATCACCACCCGGCAGCAGCGCAAACGGGTCTCCCTAGAGCCCCACCAGGGCCCTGGAAC
TCCTGAGTCTAAGAAGGCCACCAGCTGTTTCCCACGCCCCATGACTCCCCGAGACCGACATGAA
GGGCGCAAACAGAGCACTACTGAGGCCCAGAAGAAAGCAGCTCCAGCTTCTACTAAACAGGCTG
ACCGGCGCCAGTCGATGGCCTTCAGCATCCTCAACACACCCAAGAAGCTAGGGAACAGCCTTCT
GCGGCGGGGAGCCTCAAAGAAGGCCCTGTCCAAGGCTTCCCCCAACACTCGCAGTGGAACCCG
CCGTTCTCCGCGCATTGCCACCACCACAGCCAGTGCCGCCACTGCTGCCGCCATTGGTGCCACC
CCTCGAGCCAAGGGCAAGGCAAAGCACTAAAGGGCCAGTACCAGTGAGTGGCCCCACCTGTGT
CCCCGATGCTGCCGTCACCTGGTCCTCCGCCTACTGTCCCTCTCAGTGCCTTCTCTCAGCTCCCA
GGCCAACAGTAGCCAAACCCCTAGAGACAGTGATGCCTGCCCGCACCCTGGCCTGGCCCCTGG
TCCTTCACTGGCGCCTTCTCGGAGCTGGCCCAGGGGGCCTGGAGCATGGACAGTGTGGGCGCT
CTCCCTACCTTGCCTCCTTTTTTCTTAAAGCAAAGTCACTTCTCCATCACAACCAGATTTGAGGCT
GGTTTTGATGGCTGGGTCCTTGGGCCTGGCCAGTCTTCCTCTTAGCCTCTGGATCTAGAAGGGA
CCATAAGAGGAGTAGGCCCTGGTTCCTGCTGTCCTGGTGGCTGGGCCAGCAGGGGCCCTCACT
CTTGAAGTCCAGGACTGGGTCTGACCTGGTGGGAGCACCTGCCAGAGGATGCTCTTTCCCAGGA
CGGATGGGCCCTGTGTCTCAGGAGTGGGGTTGGGGGACAGCCTTCAGCAGCAGCTCACACCCT
ACCTTCCCCAGACTTGCACTGGGGTGGGATTTGGAGTGATGGGAAGGTTTTTAAGGGCCGGGGA
TGGATCTTTTCTAAATGTTATTACTTGTAAATAAAGTCTATTTTT Seq. ID No. 133

NM_004083: DNA-damage-inducible transcript 3 (DDIT3)
GGCACGAGGGAGAGAGAGAGACTTAAGTCTAAGGCACTGAGCGTATCATGTTAAAGATGAGCGG
GTGGCAGCGACAGAGCCAAAATCAGAGCTGGAACCTGAGGAGAGAGTGTTCAAGAAGGAAGTGT

ATCTTCATACATCACCACACCTGAAAGCAGATGTGCTTTTCCAGACTGATCCAACTGCAGAGATG
GCAGCTGAGTCATTGCCTTTCTCCTTTGGGACACTGTCCAGCTGGGAGCTGGAAGCCTGGTATG
AGGACCTGCAAGAGGTCCTGTCTTCAGATGAAAATGGGGGTACCTATGTTTCACCTCCTGGAAAT
GAAGAGGAAGAATCAAAAATCTTCACCACTCTTGACCCTGCTTCTCTGGCTTGGCTGACTGAGGA
GGAGCCAGAACCAGCAGAGGTCACAAGCACCTCCCAGAGCCCTCACTCTCCAGATTCCAGTCAG
AGCTCCCTGGCTCAGGAGGAAGAGGAGGAAGACCAAGGGAGAACCAGGAAACGGAAACAGAGT
GGTCATTCCCCAGCCCGGGCTGGAAAGCAGCGCATGAAGGAGAAAGAACAGGAGAATGAAAGG
AAAGTGGCACAGCTAGCTGAAGAGAATGAACGGCTCAAGCAGGAAATCGAGCGCCTGACCAGG
GAAGTAGAGGCGACTCGCCGAGCTCTGATTGACCGAATGGTGAATCTGCACCAAGCATGAACAA
TTGGGAGCATCAGTCCCCCACTTGGGCCACACTACCCACCTTTCCCAGAAGTGGCTACTGACTAC
CCTCTCACTAGTGCCAATGATGTGACCCTCAATCCCACATACGCAGGGGGAAGGCTTGGAGTAG
ACAAAAGGAAAGGTCTCAGCTTGTATATAGAGATTGTACATTTATTTATTACTGTCCCTATCTATTA
AAGTGACTTTCTATGAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAAA
Seq. ID No.134

NM_016272: Transducer of ERBB2 (TOB2)
ACTGGGGCCCACAGTCAGACATGAGCCACTGGTGGGACAGAAATAGGCTCCTGGTTCTGTGTGA
TCCANAGTTGGTGCTTTTCTGTCTATCCCTAGCTGTTGGTCACCACCAGCTTTCTGCATATTTTCT
CACGGTGCCTCTCATTTCCCAGAGCCGCCTGGAGCCCAAGGCTGTACACGTGCCCTGTGCTGAT
TCTCTGCCTAGGAAAGGACCATGCAGCTAGAGATCAAAGTGGCCCTGAACTTCATCATCTCCTAC
TTGTACAACAAGCTGCCCCGGCGCCGGGCAGACCTGTTTGGGGAGGAGCTAGAGCGGCTTTTG
AAAAAGAAATATGAAGGCCACTGGTACCCTGAGAAGCCACTGAAAGGCTCTGGCTTCCGCTGTGT
TCACATTGGGGAGATGGTGGACCCCGTGGTGGAGCTGGCCGCCAAGCGGAGTGGCCTGGCAGT
GGAAGATGTGCGGGCCAATGTGCCTGAGGAGCTGAGTGTCTGGATTGATCCCTTTGAGGTGTCC
TACCAGATTGGTGAGAAGGGAGCTGTGAAAGTGCTGTACCTGGATGACAGTGAGGGTTGCGGTG
CCCCAGAGCTGGACAAGGAGATCAAGAGCAGCTTCAACCCTGACGCCCAGGTGTTCGTGCCCAT
TGGCAGCCAGGACAGCTCCCTGTCCAACTCCCCATCGCCATCCTTTGGCCAGTCACCCAGCCCT
ACCTTCATTCCCCGCTCCGCTCAGCCCATCACCTTCACCACCGCCTCCTTCGCTGCCACCAAATT
TGGCTCCACTAAGATGAAGAAGGGGGGCGGGGCAGCAAGTGGTGGGGGTGTAGCCAGCAGTG
GGGCGGGTGGCCAGCAGCCACCACAGCAGCCTCGCATGGCCCGCTCACCCACCAACAGCCTGC
TGAAGCACAAGAGCCTCTCTCTGTCTATGCATTCACTGAACTTCATCACGGCCAACCCGGCCCCT
CAGTCCCAGCTCTCACCCAATGCCAAGGAGTTCGTGTACAACGGTGGTGGCTCACCCAGCCTCT
TCTTTGATGCGGCCGATGGCCAGGGCAGCGGCACCCCAGGCCCGTTTGGAGGCAGTGGGGCTG
GCACCTGCAACAGCAGCAGCTTTGACATGGCCCAGGTATTTGGAGGTGGTGCCAACAGCCTCTT
CCTGGAGAAGACACCCTTTGTGGAAGGCCTCAGCTACAACCTGAACACCATGCAGTATCCCAGC
CAGCAGTTCCAGCCCGTGGTGCTGGCCAACTGACCATCTACCTGCCCGTGGGGCCAGGAGCAC
CCAAGACCACAGAAAAGAGAAAGGAAAGGCCAAAAAAAAGAGGAAAAGAAAAAAAAAAAAA
Seq. ID No. 135

Amino acid sequences

NM_002982: Small inducible cytokineA2 (SCYA2)
MKVSAALLCLLLIAATFIPQGLAQPDAINAPVTCCYNFTNRKISVQRLASYRRITSSKCP
KEAVIFKTIVAKEICADPKQKWVQDSMDHLDKQTQTPKT Seq. ID No. 136

NM_015675: Growth arrest and DNA-damage-inducible beta (GADD45B)
MTLEELVACDNAAQKMQTVTAAVEELLVAAQRQDRLTVGVYESAKLMNVDPDSVVLCLLA
IDEEEEDDIALQIHFTLIQSFCCDNDINIVRVSGNARLAQLLGEPAETQGTTEARDLHCL
PFLQNPHTDAWKSHGLVEVASYCEESRGNNQWVPYISLQER Seq. ID No.137

NM_002964: S100 calcium binding protein A8 (S100A8)
MLTELEKALNSIIDVYHKYSLIKGNFHAVYRDDLKKLLETECPQYIRKKGADVWFKELDI
NTDGAVNFQEFLILVIKMGVAAHKKSHEESHKE Seq. ID No.138

NM_078467: Cyclin-dependent kinase inhibitor 1A p21/Cip1 (CDKN1A)
MSEPAGDVRQNPCGSKACRRLFGPVDSEQLSRDCDALMAGCIQEARERWNFDFVTETPLE
GDFAWERVRGLGLPKLYLPTGPRRGRDELGGGRRPGTSPALLQGTAEEDHVDLSLSCTLV
PRSGEQAEGSPGGPGDSQGRKRRQTSMTDFYHSKRRLIFSKRKP Seq. ID No.139

NM_016232: Interleukin 1receptor-like 1 (IL1RL1)
MGFWILAILTILMYSTAAKFSKQSWGLENEALIVRCPRQGKPSYTVDWYYSQTNKSIPTQ
ERNRVFASGQLLKFLPAEVADSGIYTCIVRSPTFNRTGYANVTIYKKQSDCNVPDYLMYS
TVSGSEKNSKIYCPTIDLYNWTAPLEWFKNCQALQGSRYRAHKSFLVIDNVMTEDAGDYT
CKFIHNENGANYSVTATRSFTVKDEQGFSLFPVIGAPAQNEIKEVEIGKNANLTCSACFG
KGTQFLAAVLWQLNGTKITDFGEPRIQQEEGQNQSFSNGLACLDMVLRIADVKEEDLLLQ
YDCLALNLHGLRRHTVRLSRKNPIDHHSIYCIIAVCSVFLMLINVLVIILKMFWIEATLL
WRDIAKPYKTRNDGKLYDAYVVYPRNYKSSTDGASRVEHFVHQILPDVLENKCGYTLCIY
GRDMLPGEDVVTAVETNIRKSRRHIFILTPQITHNKEFAYEQEVALHCALIQNDAKVILI
EMEALSELDMLQAEALQDSLQHLMKVQGTIKWREDHIANKRSLNSKFWKHVRYQMPVPSK
IPRKASSLTPLAAQKQ Seq. ID No.140

NM_004613: Transglutaminase 2 (TGM2)
MAEELVLERCDLELETNGRDHHTADLCREKLVVRRGQPFWLTLHFEGRNYQASVDSLTFSVVTGPAPSQEAGTKA
RFPLRDAVEEGDWTATVVDQQDCTLSLQLTTPANAPIGLYRLSLEASTGYQGSSFVLGHFILLFNAWCPADAVYL
DSEEERQEYVLTQQGFIYQGSAKFIKNIPWNFGQFQDGILDICLILLDVNPKFLKNAGRDCSRRSSPVYVGRVGS
GMVNCNDDQGVLLGRWDNNYGDGVSPMSWIGSVDILRRWKNHGCQRVKYGQCWVFAAVACTVLRCLGIPTRVVTN
YNSAHDQNSNLLIEYFRNEFGEIQGDKSEMIWNFHCWVESWMTRPDLQPGYEGWQALDPTPQEKSEGTYCCGPVP
VRAIKEGDLSTKYDAPFVFAEVNADVVDWIQQDDGSVHKSINRSLIVGLKISTKSVGRDEREDITHTYKYPEGSS
EEREAFTRANHLNKLAEKEETGMAMRIRVGQSMNMGSDFDVFAHITNNTAEEYVCRLLLCARTVSYNGILGPECG
TKYLLNLTLEPFSEKSVPLCILYEKYRDCLTESNLIKVRALLVEPVINSYLLAERDLYLENPEIKIRILGEPKQK
RKLVAEVSLQNPLPVALEGCTFTVEGAGLTEEQKTVEIPDPVEAGEEVKVRMDLVPLHMGLHKLVVNFESDKLKA
VKGFRNVIIGPA Seq ID No. 141

NM_012323: V-maf musculo aponeurotic fibrosarcoma oncogene homolog F (MAFF)
MSVDPLSSKALKIKRELSENTPHLSDEALMGLSVRELNRHLRGLSAEEVTRLKQRRRTLK
NRGYAASCRVKRVCQKEELQKQKSELEREVDKLARENAAMRLELDALRGKCEALQGFARS
VAAARGPATLVAPASVITIVKSTPGSGSGPAHGPDPAHGPASCS Seq. ID No. 142

NM_001085: Serine or cysteine proteinase inhibitor clade A member 3 (SERPINA3)
MERMLPLLALGLLAAGFCPAVLCHPNSPLDEENLTQENQDRGTHVDLGLASANVDFAFSL
YKQLVLKALDKNVIFSPLSISTALAFLSLGAHNTTLTEILKASSSPHGDLLRQKFTQSFQ

HLRAPSISSSDELQLSMGNAMFVKEQLSLLDRFTEDAKRLYGSEAFATDFQDSAAAKKLI
NDYVKNGTRGKITDLIKDPDSQTMMVLVNYIFFKAKWEMPFDPQDTHQSRFYLSKKKWVM
VPMMSLHHLTIPYFRDEELSCTVVELKYTGNASALFILPDQDKMEEVEAMLLPETLKRWR
DSLEFREIGELYLPKFSISRDYNLNDILLQLGIEEAFTSKADLSGITGARNLAVSQVVHK
VVSDVFEEGTEASAATAVKITLLSALVETRTIVRFNRPFLMIIVPTDTQNIFFMSKVTNP
SKPRACIKQWGSQ Seq. ID No.143

NM_001511: GRO1 oncogene melanoma growth stimulating activityalpha (GRO1)
MARAALSAAPSNPRLLRVALLLLLLVAAGRRAAGASVATELRCQCLQTLQGIHPKNIQSV
NVKSPGPHCAQTEVIATLKNGRKACLNPASPIVKKIIEKMLNSDKSN Seq. ID No.144

NM_000591: CD14 antigen (CD14)
MERASCLLLLLLPLVHVSATTPEPCELDDEDFRCVCNFSEPQPDWSEAFQCVSAVEVEIH
AGGLNLEPFLKRVDADADPRQYADTVKALRVRRLTVGAAQVPAQLLVGALRVLAYSRLKE
LTLEDLKITGTMPPLPLEATGLALSSLRLRNVSWATGRSWLAELQQWLKPGLKVLSIAQA
HSPAFSCEQVRAFPALTSLDLSDNPGLGERGLMAALCPHKFPAIQNLALRNTGMETPTGV
CAALAAAGVQPHSLDLSHNSLRATVNPSAPRCMWSSALNSLNLSFAGLEQVPKGLPAKLR
VLDLSCNRLNRAPQPDELPEVDNLTLDGNPFLVPGTALPHEGSMNSGVVPACARSTLSVG
VSGTLVLLQGARGFA Seq. ID No.145

NM_015319: Tensin 2 (KIAA1075)
MDGGGVCVGRGDLLSSPQALGQLLRKESRPRRAMKPRKAEPHSFREKVFRKKPPVCAVCK
VTIDGTGVSCRVCKVATHRKCEAKVTSACQALPPVELRRNTAPVRRIEHLGSTKSLNHSK
QRSTLPRSFSLDPLMERRWDLDLTYVTERILAAAFPARPDEQRHRGHLRELAHVLQSKHR
DKYLLFNLSEKRHDLTRLNPKVQDFGWPELHAPPLDKLCSICKAMETWLSADPQHVVVLY
CKGNKGKLGVIVSAYMHYSKISAGADQALATLTMRKFCEDKVATELQPSQRRYISYFSGL
LSGSIRMNSSPLFLHYVLIPMLPAFEPGTGFQPFLKIYQSMQLVYTSGVYHIAGPGPQQL
CISLEPALLLKGDVMVTCYHKGGRGTDRTLVFRVQFHTCTIHGPQLTFPKDQLDEAWTDE
RFPFQASVEFVFSSSPEKIKGSTPRNDPSVSVDYNTTEPAVRWDSYENFNQHHEDSVDGS
LTHTRGPLDGSPYAQVQRPPRQTPPAPSPEPPPPPMLSVSSDSGHSSTLTTEPAAESPGR
PPPTAAERQELDRLLGGCGVASGGRGAGRETAILDDEEQPTVGGGPHLGVYPGHRPGLSR
HCSCRQGYREPCGVPNGGYYRPEGTLERRRLAYGGYEGSPQGYAEASMEKRRLCRSLSEG
LYPYPPEMGKPATGDFGYRAPGYREVVILEDPGLPALYPCPACEEKLALPTAALYGLRLE
REAGEGWASEAGKPLLHPVRPGHPLPLLLPACGHHHAPMPDYSCLKPPKAGEEGHEGCSY
TMCPEGRYGHPGYPALVTYSYGGAVPSYCPAYGRVPHSCGSPGEGRGYPSPGAHSPRAGS
ISPGSPPYPQSRKLSYEIPTEEGGDRYPLPGHLASAGPLASAESLEPVSWREGPSGHSTL
PRSPRDAPCSASSELSGPSTPLHTSSPVQGKESTRRQDTRSPTSAPTQRLSPGEALPPVS
QAGTGKAPELPSGSGPEPLAPSPVSPTFPPSSPSDWPQERSPGGHSDGASPRSPVPTTLP
GLRHAPWQGPRGPPDSPDGSPLTPVPSQMPWLVASPEPPQSSPTPAFPLAASYDTNGLSQ
PPLPEKRHLPGPGQQPGPWGPEQASSPARGISHHVTFAPLLSDNVPQTPEPPTQESQSNV
KFVQDTSKFWYKPHLSRDQAIALLKDKDPGAFLIRDSHSFQGAYGLALKVATPPPSAQPW
KGDPVEQLVRHFLIETGPKGVKIKGCPSEPYFGSLSALVSQHSISPISLPCCLRILSKDP
LEETPEAPVPTNMSTAADLLRQGAACSVLYLTSVETESLTGPQAVARASSAALSCSPRPT
PAVVHFKVSAQGITLTDNQRKLFFRRHYPVNSITFSSTDPQDRRWTNPDGTTSKIFGFVA
KKPGSPWENVCHLFAELDPDQPAGAIVTFITKVLLGQRK Seq. ID No. 146

NM_001276: Chitinase 3-like 1, cartilage glycoprotein-39 (CHI3L1)
MGVKASQTGFVVLVLLQCCSAYKLVCYYTSWSQYREGDGSCFPDALDRFLCTHIIYSFAN
ISNDHIDTWEWNDVTLYGMLNTLKNRNPNLKTLLSVGGWNFGSQRFSKIASNTQSRRTFI
KSVPPFLRTHGFDGLDLAWLYPGRRDKQHFTTLIKEMKAEFIKEAQPGKKQLLLSAALSA
GKVTIDSSYDIAKISQHLDFISIMTYDFHGAWRGTTGHHSPLFRGQEDASPDRFSNTDYA
VGYMLRLGAPASKLVMGIPTFGRSFTLASSETGVGAPISGPGIPGRFTKEAGTLAYYEIC
DFLRGATVHRTLGQQVPYATKGNQWVGYDDQESVKSKVQYLKDRQLAGAMVWALDLDDFQ
GSFCGQDLRFPLTNAIKDALAAT Seq. ID No.147

NM_004353: Serine or cysteine proteinase inhibitor clade H (SERPINH1)
MRSLLLGTLCLLAVALAAEVKKPVEAAAPGTAEKLSSKATTLAEPSTGLAFSLYQAMAKD
QAVENILVSPVVVASSLGLVSLGGKATTASQAKAVLSAEQLRDEEVHAGLGELLRSLSNS
TARNVTWKLGSRLYGPSSVSFADDFVRSSKQHYNCEHSKINFPDKRSALQSINEWAAQTT
DGKLPEVTKDVERTDGALLVNAMFFKPHWDEKFHHKMVDNRGFMVTRSYTVGVTMMHRTG
LYNYYDDEKEKLQLVEMPLAHKLSSLIILMPHHVEPLERLEKLLTKEQLKIWMGKMQKKA
VAISLPKGVVEVTHDLQKHLAGLGLTEAIDKNKADLSRMSGKKDLYLASVFHATAFELDT
DGNPFDQDIYGREELRSPKLFYADHPFIFLVRDTQSGSLLFIGRLVRLKGDKMRDEL Seq. ID No.148

NM_005952: Metallothionein 1X(MT1X)
MDPNCSCSPVGSCACAGSCKCKECKCTSCKKSCCSCCPVGCAKCAQGCICKGTSDKCSCCA Seq. ID No.149

XM_030707: KIAA0620 protein (KIAA0620)
MAPRAAGGAPLSARAAAASPPPFQTPPRCPVPLLLLLLLGAARAGALEIQRRFPSPTPTN
NFALDGAAGTVYLAAVNRLYQLSGANLSLEAEAAVGPVPDSPLCHAPQLPQASCEHPRRL
TDNYNKILQLDPGQGLVVVCGSIYQGFCQLRRRGNISAVAVRFPPAAPPAEPVTVFPSML
NVAANHPNASTVGLVLPPAAGAGGSRLLVGATYTGYGSSFFPRNRSLEDHRFENTPEIAI
RSLDTRGDLAKLFTFDLNPSDDNILKIKQGAKEQHKLGFVSAFLHPSDPPPGAQSYAYLA
LNSEARAGDKESQARSLLARICLPHGAGGDAKKLTESYIQLGLQCAGGAGRGDLYSRLVS
VFPARERLFAVFERPQGSPAARAAPAALCAFRFADVRAAIRAARTACFVEPAPDVVAVLD
SVVQGTGPACERKLNIQLQPEQLDCGAAHLQHPLSILQPLKATPVFRAPGLTSVAVASVN
NYTAVFLGTVNGRLLKINLNESMQVVSRRVVTVAYGEPVHHVMQFDPADSGYLYLMTSHQ
MARVKVAACNVHSTCGDCVGAADAYCGWCALETRCTLQQDCTNSSQQHFWTSASEGPSRC
PAMTVLPSEIDVRQEYPGMILQISGSLPSLSGMEMACDYGNNIRTVARVPGPAFGHQIAY
CNLLPRDQFPPFPPNQDHVTVEMSVRVNGRNIVKANFTIYDCSRTAQVYPHTACTSCLSA
QWPCFWCSQQHSCVSNQSRCEASPNPTSPQDCPRTLLSPLAPVPTGGSQNILVPLANTAF
FQGAALECSFGLEEIFEAVWVNESVVRCDQVVLHTTRKSQVFPLSLQLKGRPARFLDSPE
PMTVMVYNCAMGSPDCSQCLGREDLGHLCVWSDGCRLRGPLQPMAGTCPAPEIRAIEPLS
GPLDGGTLLTIRGRNLGRRLSDVAHGVWIGGVACEPLPDRYTVSEEIVCVTGPAPGPLSG
VVTVNASKEGKSRDRFSYVLPLVHSLEPTMGPKAGGTRITIHGNDLHVGSELQVLVNDTD
PCTELMRTDTSIACTMPEGALPAPVPVCVRFERRGCVHGNLTFWYMQNPVITAISPRRSP
VSGGRTITVAGERFHMVQNVSMAVHHIGREPTLCKVLNSTLITCPSPGALSNASAPVDFF
INGRAYADEVAVAEELLDPEEAQRGSRFRLDYLPNPQFSTAKREKWIKHHPGEPLTLVIH
KEQDSLGLQSHEYRVKIGQVSCDIQIVSDRIIHCSVNESLGAAVGQLPITIQVGNFNQTI
ATLQLGGSETAIIVSIVICSVLLLLSVVALFVFCTKSRRAERYWQKTLLQMEEMESQIRE
EIRKGFAELQTDMTDLTKELNRSQGIPFLEYKHFVTRTFFPKCSSLYEERYVLPSQTLNS
QGSSQAQETHPLLGEWKIPESCRPNMEEGISLFSSLLNNKHFLIVFVHALEQQKDFAVRD
RCSLASLLTIALHGKLEYYTSIMKELLVDLIDASAAKNPKLMLRRTESVVEKMLTNWMSI
CMYSCLRETVGEPFFLLLCAIKQQINKGSIDAITGKARYTLSEEWLLRENIEAKPRNLNV
SFQGCGMDSLSVRAMDTDTLTQVKEKILEAFCKNVPYSQWPRAEDVDLGGSPPAHRATSF
GTWTTPQWWKTAARSLTRWPITRSLKVPPWP Seq. ID No.150

NM_003254: Tissue inhibitor of metalloproteinase 1 (TIMP1)
MAPFEPLASGILLLLWLIAPSRACTCVPPHPQTAFCNSDLVIRAKFVGTPEVNQTTLYQR
YEIKMTKMYKGFQALGDAADIRFVYTPAMESVCGYFHRSHNRSEEFLIAGKLQDGLLHIT
TCSFVAPWNSLSLAQRRGFTKTYTVGCEECTVFPCLSIPCKLQSGTHCLWTDQLLQGSEK
GFQSRHLACLPREPGLCTWQSLRSQIA Seq. ID No.151

NM_006185: Nuclear mitotic apparatus protein 1 (NUMA1)
MTLHATRGAALLSWVNSLHVADPVEAVLQLQDCSIFIKIIDRIHGTEEGQQILKQPVSER
LDFVCSFLQKNRKHPSSPECLVSAQKVLEGSELELAKMTMLLLYHSTMSSKSPRDWEQFE
YKIQAELAVILKFVLDHEDGLNLNEDLENFLQKAPVPSTCSSTFPEELSPPSHQAKREIR
FLELQKVASSSSGNNFLSGSPASPMGDILQTPQFQMRRLKKQLADERSNRDELELELAEN
RKLLTEKDAQIAMMQQRIDRLALLNEKQAASPLEPKELEELRDKNESLTMRLHETLKQCQ
DLKTEKSQMDRKINQLSEENGDLSFKLREFASHLQQLQDALNELTEEHSKATQEWLEKQA

QLEKELSAALQDKKCLEEKNEILQGKLSQLEEHLSQLQDNPPQEKGEVLGDVLQLETLKQ
EAATLAANNTQLQARVEMLETERGQQEAKLLAERGHFEEEKQQLSSLITDLQSSISNLSQ
AKEELEQASQAHGARLTAQVASLTSELTTLNATIQQQDQELAGLKQQAKEKQAQLAQTLQ
QQEQASQGLRHQVEQLSSSLKQKEQQLKEVAEKQEATRQDHAQQLATAAEEREASLRERD
AALKQLEALEKEKAAKLEILQQQLQVANEARDSAQTSVTQAQREKAELSRKVEELQACVE
TARQEQHEAQAQVAELELQLRSEQQKATEKERVAQEKDQLQEQLQALKESLKVTKGSLEE
EKRRAADALEEQQRCISELKAETRSLVEQHKRERKELEEERAGRKGLEARLLQLGEAHQA
ETEVLRRELAEAMAAQHTAESECEQLVKEVAAWRDGYEDSQQEEAQYGAMFQEQLMTLKE
ECEKARQELQEAKEKVAGIESHSELQISRQQNKLAELHANLARALQQVQEKEVRAQKLAD
DLSTLQEKMAATSKEVARLETLVRKAGEQQETASRELVKEPARAGDRQPEWLEEQQGRQF
CSTQAALQAMEREAEQMGNELERLRAALMESQGQQQEERGQQEREVARLTQERGRAQADL
ALEKAARAELEMRLQNALNEQRVEFATLQEALAHALTEKEGKDQELAKLRGLEAAQIKEL
EELRQTVKQLKEQLAKKEKEHASGSGAQSEAAGRTEPTGPKLEALRAEVSKLEQQCQKQQ
EQADSLERSLEAERASRAERDSALETLQGQLEEKAQELGHSQSALASAQRELAAFRTKVQ
DHSKAEDEWKAQVARGRQEAERKNSLISSLEEEVSILNRQVLEKEGESKELKRLVMAESE
KSQKLEESCACCRQRQPATVPELQNAALLCGRRCRASGREAEKQRVASENLRQELTSQAE
RAEELGQELKAWQEKFFQKEQALSTLQLEHTSTQALVSELLPAKHLCQQLQAEQAAAEKR
HREELEQSKQAAGGLRAELLRAQRELGELIPLRQKVAEQERTAQQLRAEKASYAEQLSML
KKAHGLLAEENRGLGERANLGRQFLEVELDQAREKYVQELAAVRADAETRLAEVQREAQS
TARELEVMTAKYEGAKVKVLEERQRFQEERQKLTAQVEELSKKLADSDQASKVQQQKLKA
VQAQGGESQQEAQRFQAQLNELQAQLSQKEQAAEHYKLQMEKAKTHYDAKKQQNQELQEQ
LRSLEQLQKENKELRAEAERLGHELQQAGLKTKEAEQTCRHLTAQVRSLEAQVAHADQQL
RDLGKFQVATDALKSREPQAKPQLDLSIDSLDLSCEEGTPLSITSKLPRTQPDGTSVPGE
PASPISQRLPPKVESLESLYFTPIPARSQAPLESSLDSLGDVFLDSGRKTRSARRRTTQI
INITMTKKLDVEEPDSANSSFYSTRSAPASQASLRATSSTQSLARLGSPDYGNSALLSLP
GYRPTTRSSARRSQAGVSSGAPPGRNSFYMGTCQDEPEQLDDWNRIAELQQRNRVCPPHL
KTCYPLESRPSLSLGTITDEEMKTGDPQETLRRASMQPIQIAEGTGITTRQQRKRVSLEP
HQGPGTPESKKATSCFPRPMTPRDRHEGRKQSTTEAQKKAAPASTKQADRRQSMAFSILN
TPKKLGNSLLRRGASKKALSKASPNTRSGTRRSPRIATTTASAATAAAIGATPRAKGKAK
H Seq. ID No.152

NM_004083: DNA-damage-inducible transcript 3 (DDIT3)
MAAESLPFSFGTLSSWELEAWYEDLQEVLSSDENGGTYVSPPGNEEEESKIFTTLDPASL
AWLTEEEPEPAEVTSTSQSPHSPDSSQSSLAQEEEEEDQGRTRKRKQSGHSPARAGKQRM
KEKEQENERKVAQLAEENERLKQEIERLTREVEATRRALIDRMVNLHQA Seq. ID No.153

NM_016272 Transducer of ERBB2 (TOB2)
MQLEIKVALNFIISYLYNKLPRRRADLFGEELERLLKKKYEGHWYPEKPLKGSGFRCVHI
GEMVDPVVELAAKRSGLAVEDVRANVPEELSVWIDPFEVSYQIGEKGAVKVLYLDDSEGC
GAPELDKEIKSSFNPDAQVFVPIGSQDSSLSNSPSPSFGQSPSPTFIPRSAQPITFTTAS
FAATKFGSTKMKKGGGAASGGGVASSGAGGQQPPQQPRMARSPTNSLLKHKSLSLSMHSL
NFITANPAPQSQLSPNAKEFVYNGGGSPSLFFDAADGQGSGTPGPFGGSGAGTCNSSSFD
MAQVFGGGANSLFLEKTPFVEGLSYNLNTMQYPSQQFQPVVLAN Seq. ID No.154

The foregoing description illustrates preferred embodiments of the present invention. It should be understood that those skilled in the art will envision modifications of the embodiments that are covered by the following claims.

WHAT IS CLAIMED IS:

1. A method of screening for schizophrenia in a population comprising determining the magnitude of expression, in members of the population, of at least one gene selected from the group consisting of those disclosed in Table 1 in a sample and comparing the magnitude of expression to a baseline magnitude of expression of the gene, wherein increased gene expression indicates the presence of schizophrenia.

2. A method of screening for schizophrenia in a population according to claim 1 wherein the sample is taken from brain, spinal cord, lymphatic fluid, blood, urine or feces.

3. A method of screening for schizophrenia in a population according to claim 2 wherein the sample is taken from the anterior cingulate.

4. A method of screening for schizophrenia in a population according to claim 1 wherein the population is human.

5. A method for diagnosing schizophrenia in a host comprising determining the magnitude of expression of at least one gene selected from the group consisting of those disclosed in Table 1 in a sample and comparing the magnitude of expression to a baseline magnitude of expression of the gene, wherein increased gene expression indicates the presence of schizophrenia.

6. A method for diagnosing schizophrenia in a host according to claim 5 wherein the sample is taken from brain, spinal cord, lymphatic fluid, blood, urine or feces.

7. A method for diagnosing schizophrenia in a host according to claim 6 wherein the sample is taken from the anterior cingulate.

8. A method for diagnosing schizophrenia in a host according to claim 5 wherein the host is human.

9. A method for treating schizophrenia in a host comprising lowering expression of at least one gene selected from the group consisting of those disclosed in Table 1 by administering to the host an expression lowering amount of antisense oligonucleotide.

10. A method for treating schizophrenia in a host according to claim 9 wherein the host is human.

11. A method for treating schizophrenia in a host comprising lowering expression of at least one gene selected from the group consisting of those disclosed in Table 1 by administering to the host an expression lowering amount of a ribozyme which cleaves RNA associated with expression of the gene.

12. A method for treating schizophrenia in a host according to claim 11 wherein the host is human.

13. A method for treating schizophrenia in a host comprising lowering expression of a gene selected from the group consisting of those disclosed in Table 1 by administering one or more nucleic acid molecules designed to promote triple helix formation with said gene .

14. A method for treating schizophrenia in a host according to claim 13 wherein the host is human.

15. A method for treating schizophrenia is in a host comprising reducing the amount of at least one protein selected from the group consisting of those encoded by the genes disclosed in Table 1 in a patient by administering an effective amount of antibody or functional antibody fragment sufficient to interfere with the normal activity of the protein.

16. A method for treating schizophrenia in a host according to claim 15 wherein the host is human.

17. A method for treating schizophrenia in a host according to claim 15 wherein the antibody or functional antibody fragment is selected from the group consisting of whole antibody, humanized antibody, chimeric antibody, Fab fragment, Fab' fragment, $F(ab')_2$ fragment, single chain Fv fragment and diabody.

18. A transgenic nonhuman animal comprising stably integrated in its genome an increased copy number of a gene selected from the group consisting of the genes disclosed in Table 1) wherein said gene is expressed at higher than baseline levels and the animal exhibits schizophrenic behavior.

19. A transgenic nonhuman animal according to claim 18 wherein the transgenic nonhuman animal is a mammal.

20. A transgenic nonhuman animal comprising stably integrated in its genome a gene selected from the group consisting of those disclosed in Table 1 , wherein expression of the gene is enhanced by one or more alterations in regulatory sequences of the gene such that the gene is expressed at higher than baseline levels and the animal exhibits schizophrenic behavior.

21. A transgenic nonhuman animal according to claim 20 wherein the transgenic nonhuman animal is a mammal.

22. A transgenic nonhuman animal according to claim 20 wherein the one or more alterations comprises substitution of a promoter having a higher rate of expression than the native promoter of the gene.

23. A transgenic nonhuman animal according to claim 22 wherein the promoter is an inducible promoter.

24. A transgenic nonhuman knockout animal whose genome comprises a homozygous disruption in one or more genes selected from the group consisting of those disclosed in Table 1 wherein said homozygous disruption prevents the expression of the gene, and wherein said homozygous disruption results in the transgenic knockout animal exhibiting decreased expression levels of the one or more genes as compared to a wild-type animal.

25. A method of screening for a therapeutic agent that modulates symptoms of schizophrenia comprising administering a candidate compound to a transgenic nonhuman animal according to claim 18 and determining the effect of the compound on symptoms associated with schizophrenia.

26. A method of screening for a therapeutic agent that modulates symptoms of schizophrenia comprising combining a candidate compound with a transgenic nonhuman animal according to claim 20 and determining the effect of the compound on symptoms associated with schizophrenia.

27. A method of screening for a therapeutic agent that modulates symptoms of schizophrenia comprising combining a candidate compound with a transgenic nonhuman animal according to claim 24 and determining the effect of the compound on symptoms associated with schizophrenia.

28. A method of screening for a compound useful in the treatment of schizophrenia comprising operatively linking a reporter gene which expresses a detectable protein to a regulatory sequence for a gene selected from the group consisting of those disclosed in Table 1 to produce a reporter construct; transfecting a cell with the reporter construct; exposing the transfected cell to a test compound; and comparing the level of expression of the reporter gene after exposure to the test compound to the level of expression before exposure to the test compound, wherein

a lower level of expression after exposure is indicative of a compound useful for the treatment of schizophrenia.

29. A method for treating schizophrenia in a host comprising lowering expression of a gene selected from the group consisting of those disclosed in Table 1 by administering one or more RNAi molecules designed to inhibit the expression of said gene .

30. A method for treating schizophrenia in a host according to claim 29 wherein the host is human.

ABSTRACT

The genes encoding SCYA2, GADD45B, S100A8, CDKN1A, IL1RL1, TGM2, MAFF, SERPINA3, GRO1, CD14, KIAA1075, CHI3L1, SERPINH1, MT1X, KIAA0620, TIMP1, NUMA1, DDIT3 and TOB2 , are upregulated in the anterior cingulate of schizophrenic patients compared to normal patients and as such are useful drug targets for schizophrenia. Methods of screening, diagnosing and treating schizophrenia based on these genes are provided. Transgenic nonhuman animals having increased copy number or increased expression levels of these genes are also provided. The transgenic nonhuman animals are used in methods for screening for potential therapeutic agents.

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<110> BUXTON, Francis Paul
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CARPENTER, William Twitty

<120> METHODS FOR DIAGNOSING AND TREATING SCHIZOPHRENIA

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gcagcgcatg aaggagaaag aacaggagaa tgaaaggaaa gtggcacagc tagctgaaga 600

gaatgaacgg ctcaagcagg aaatcgagcg cctgaccagg gaagtagagg cgactcgccg 660

agctctgatt gaccgaatgg tgaatctgca ccaagcatga acaattggga gcatcagtcc 720

cccacttggg ccacactacc cacctttccc agaagtggct actgactacc ctctcactag 780

tgccaatgat gtgaccctca atcccacata cgcaggggga aggcttggag tagacaaaag 840

gaaaggtctc agcttgtata tagagattgt acatttattt attactgtcc ctatctatta 900

aagtgacttt ctatgaaaaa aaaaaaaaaa aaaaaaaaaa aaaaaaaaaa aaaaaaaaaa 960

aaaaa 965

<210> 135
<211> 1341
<212> DNA
<213> Homo sapiens

<220>
<221> misc_feature
<222> (69)..(69)
<223> n is a, c, g, or t

<400> 135
actggggccc acagtcagac atgagccact ggtgggacag aaataggctc ctggttctgt 60

gtgatccana gttggtgctt ttctgtctat ccctagctgt tggtcaccac cagctttctg 120

catattttct cacggtgcct ctcatttccc agagccgcct ggagcccaag gctgtacacg 180

tgccctgtgc tgattctctg cctaggaaag gaccatgcag ctagagatca aagtggccct 240

gaacttcatc atctcctact tgtacaacaa gctgccccgg cgccgggcag acctgtttgg 300

ggaggagcta gagcggcttt tgaaaaagaa atatgaaggc cactggtacc ctgagaagcc 360

actgaaaggc tctggcttcc gctgtgttca cattggggag atggtggacc ccgtggtgga 420

gctggccgcc aagcggagtg gcctggcagt ggaagatgtg cgggccaatg tgcctgagga 480

gctgagtgtc tggattgatc cctttgaggt gtcctaccag attggtgaga agggagctgt 540

gaaagtgctg tacctggatg acagtgaggg ttgcggtgcc ccagagctgg acaaggagat 600

caagagcagc ttcaaccctg acgcccaggt gttcgtgccc attggcagcc aggacagctc 660

cctgtccaac tccccatcgc catcctttgg ccagtcaccc agccctacct tcattccccg 720

ctccgctcag cccatcacct tcaccaccgc ctccttcgct gccaccaaat ttggctccac 780

taagatgaag aaggggggcg gggcagcaag tggtgggggt gtagccagca gtggggcggg 840

tggccagcag ccaccacagc agcctcgcat ggcccgctca cccaccaaca gcctgctgaa 900

gcacaagagc ctctctctgt ctatgcattc actgaacttc atcacggcca acccggcccc 960

tcagtcccag ctctcaccca atgccaagga gttcgtgtac aacggtggtg gctcacccag 1020

cctcttcttt gatgcggccg atggccaggg cagcggcacc ccaggcccgt ttggaggcag 1080

tggggctggc acctgcaaca gcagcagctt tgacatggcc caggtatttg gaggtggtgc 1140

caacagcctc ttcctggaga agacaccctt tgtggaaggc ctcagctaca acctgaacac 1200

catgcagtat cccagccagc agttccagcc cgtggtgctg gccaactgac catctacctg 1260

cccgtggggc caggagcacc caagaccaca gaaaagagaa aggaaaggcc aaaaaaaaga 1320

ggaaaagaaa aaaaaaaaaa a 1341

<210> 136
<211> 99
<212> PRT
<213> Homo sapiens

<400> 136

Met Lys Val Ser Ala Ala Leu Leu Cys Leu Leu Leu Ile Ala Ala Thr
1 5 10 15

Phe Ile Pro Gln Gly Leu Ala Gln Pro Asp Ala Ile Asn Ala Pro Val
20 25 30

Thr Cys Cys Tyr Asn Phe Thr Asn Arg Lys Ile Ser Val Gln Arg Leu
35 40 45

Ala Ser Tyr Arg Arg Ile Thr Ser Ser Lys Cys Pro Lys Glu Ala Val
50 55 60

Ile Phe Lys Thr Ile Val Ala Lys Glu Ile Cys Ala Asp Pro Lys Gln
65 70 75 80

Lys Trp Val Gln Asp Ser Met Asp His Leu Asp Lys Gln Thr Gln Thr
85 90 95

Pro Lys Thr

<210> 137
<211> 161
<212> PRT
<213> Homo sapiens

<400> 137

Met Thr Leu Glu Glu Leu Val Ala Cys Asp Asn Ala Ala Gln Lys Met

1 5 10 15

Gln Thr Val Thr Ala Ala Val Glu Glu Leu Leu Val Ala Ala Gln Arg
20 25 30

Gln Asp Arg Leu Thr Val Gly Val Tyr Glu Ser Ala Lys Leu Met Asn
35 40 45

Val Asp Pro Asp Ser Val Val Leu Cys Leu Leu Ala Ile Asp Glu Glu
50 55 60

Glu Glu Asp Asp Ile Ala Leu Gln Ile His Phe Thr Leu Ile Gln Ser
65 70 75 80

Phe Cys Cys Asp Asn Asp Ile Asn Ile Val Arg Val Ser Gly Asn Ala
85 90 95

Arg Leu Ala Gln Leu Leu Gly Glu Pro Ala Glu Thr Gln Gly Thr Thr
100 105 110

Glu Ala Arg Asp Leu His Cys Leu Pro Phe Leu Gln Asn Pro His Thr
115 120 125

Asp Ala Trp Lys Ser His Gly Leu Val Glu Val Ala Ser Tyr Cys Glu
130 135 140

Glu Ser Arg Gly Asn Asn Gln Trp Val Pro Tyr Ile Ser Leu Gln Glu
145 150 155 160

Arg

<210> 138
<211> 93
<212> PRT
<213> Homo sapiens

<400> 138

Met Leu Thr Glu Leu Glu Lys Ala Leu Asn Ser Ile Ile Asp Val Tyr
1 5 10 15

His Lys Tyr Ser Leu Ile Lys Gly Asn Phe His Ala Val Tyr Arg Asp
20 25 30

Asp Leu Lys Lys Leu Leu Glu Thr Glu Cys Pro Gln Tyr Ile Arg Lys
35 40 45

Lys Gly Ala Asp Val Trp Phe Lys Glu Leu Asp Ile Asn Thr Asp Gly
50 55 60

Ala Val Asn Phe Gln Glu Phe Leu Ile Leu Val Ile Lys Met Gly Val
65 70 75 80

```
Ala Ala His Lys Lys Ser His Glu Glu Ser His Lys Glu
                85                  90
```

<210> 139
<211> 164
<212> PRT
<213> Homo sapiens

<400> 139

```
Met Ser Glu Pro Ala Gly Asp Val Arg Gln Asn Pro Cys Gly Ser Lys
1               5                   10                  15

Ala Cys Arg Arg Leu Phe Gly Pro Val Asp Ser Glu Gln Leu Ser Arg
            20                  25                  30

Asp Cys Asp Ala Leu Met Ala Gly Cys Ile Gln Glu Ala Arg Glu Arg
        35                  40                  45

Trp Asn Phe Asp Phe Val Thr Glu Thr Pro Leu Glu Gly Asp Phe Ala
    50                  55                  60

Trp Glu Arg Val Arg Gly Leu Gly Leu Pro Lys Leu Tyr Leu Pro Thr
65                  70                  75                  80

Gly Pro Arg Arg Gly Arg Asp Glu Leu Gly Gly Gly Arg Arg Pro Gly
                85                  90                  95

Thr Ser Pro Ala Leu Leu Gln Gly Thr Ala Glu Glu Asp His Val Asp
            100                 105                 110

Leu Ser Leu Ser Cys Thr Leu Val Pro Arg Ser Gly Glu Gln Ala Glu
        115                 120                 125

Gly Ser Pro Gly Gly Pro Gly Asp Ser Gln Gly Arg Lys Arg Arg Gln
    130                 135                 140

Thr Ser Met Thr Asp Phe Tyr His Ser Lys Arg Arg Leu Ile Phe Ser
145                 150                 155                 160

Lys Arg Lys Pro
```

<210> 140
<211> 556
<212> PRT
<213> Homo sapiens

<400> 140

```
Met Gly Phe Trp Ile Leu Ala Ile Leu Thr Ile Leu Met Tyr Ser Thr
1               5                   10                  15
```

```
Ala Ala Lys Phe Ser Lys Gln Ser Trp Gly Leu Glu Asn Glu Ala Leu
            20                  25                  30

Ile Val Arg Cys Pro Arg Gln Gly Lys Pro Ser Tyr Thr Val Asp Trp
        35                  40                  45

Tyr Tyr Ser Gln Thr Asn Lys Ser Ile Pro Thr Gln Glu Arg Asn Arg
    50                  55                  60

Val Phe Ala Ser Gly Gln Leu Leu Lys Phe Leu Pro Ala Glu Val Ala
65                  70                  75                  80

Asp Ser Gly Ile Tyr Thr Cys Ile Val Arg Ser Pro Thr Phe Asn Arg
                85                  90                  95

Thr Gly Tyr Ala Asn Val Thr Ile Tyr Lys Lys Gln Ser Asp Cys Asn
            100                 105                 110

Val Pro Asp Tyr Leu Met Tyr Ser Thr Val Ser Gly Ser Glu Lys Asn
        115                 120                 125

Ser Lys Ile Tyr Cys Pro Thr Ile Asp Leu Tyr Asn Trp Thr Ala Pro
    130                 135                 140

Leu Glu Trp Phe Lys Asn Cys Gln Ala Leu Gln Gly Ser Arg Tyr Arg
145                 150                 155                 160

Ala His Lys Ser Phe Leu Val Ile Asp Asn Val Met Thr Glu Asp Ala
                165                 170                 175

Gly Asp Tyr Thr Cys Lys Phe Ile His Asn Glu Asn Gly Ala Asn Tyr
            180                 185                 190

Ser Val Thr Ala Thr Arg Ser Phe Thr Val Lys Asp Glu Gln Gly Phe
        195                 200                 205

Ser Leu Phe Pro Val Ile Gly Ala Pro Ala Gln Asn Glu Ile Lys Glu
    210                 215                 220

Val Glu Ile Gly Lys Asn Ala Asn Leu Thr Cys Ser Ala Cys Phe Gly
225                 230                 235                 240

Lys Gly Thr Gln Phe Leu Ala Ala Val Leu Trp Gln Leu Asn Gly Thr
                245                 250                 255

Lys Ile Thr Asp Phe Gly Glu Pro Arg Ile Gln Gln Glu Glu Gly Gln
            260                 265                 270

Asn Gln Ser Phe Ser Asn Gly Leu Ala Cys Leu Asp Met Val Leu Arg
        275                 280                 285
```

```
Ile Ala Asp Val Lys Glu Glu Asp Leu Leu Leu Gln Tyr Asp Cys Leu
    290                 295                 300

Ala Leu Asn Leu His Gly Leu Arg Arg His Thr Val Arg Leu Ser Arg
305                 310                 315                 320

Lys Asn Pro Ile Asp His His Ser Ile Tyr Cys Ile Ile Ala Val Cys
                325                 330                 335

Ser Val Phe Leu Met Leu Ile Asn Val Leu Val Ile Ile Leu Lys Met
            340                 345                 350

Phe Trp Ile Glu Ala Thr Leu Leu Trp Arg Asp Ile Ala Lys Pro Tyr
        355                 360                 365

Lys Thr Arg Asn Asp Gly Lys Leu Tyr Asp Ala Tyr Val Val Tyr Pro
    370                 375                 380

Arg Asn Tyr Lys Ser Ser Thr Asp Gly Ala Ser Arg Val Glu His Phe
385                 390                 395                 400

Val His Gln Ile Leu Pro Asp Val Leu Glu Asn Lys Cys Gly Tyr Thr
                405                 410                 415

Leu Cys Ile Tyr Gly Arg Asp Met Leu Pro Gly Glu Asp Val Val Thr
            420                 425                 430

Ala Val Glu Thr Asn Ile Arg Lys Ser Arg Arg His Ile Phe Ile Leu
        435                 440                 445

Thr Pro Gln Ile Thr His Asn Lys Glu Phe Ala Tyr Glu Gln Glu Val
    450                 455                 460

Ala Leu His Cys Ala Leu Ile Gln Asn Asp Ala Lys Val Ile Leu Ile
465                 470                 475                 480

Glu Met Glu Ala Leu Ser Glu Leu Asp Met Leu Gln Ala Glu Ala Leu
                485                 490                 495

Gln Asp Ser Leu Gln His Leu Met Lys Val Gln Gly Thr Ile Lys Trp
            500                 505                 510

Arg Glu Asp His Ile Ala Asn Lys Arg Ser Leu Asn Ser Lys Phe Trp
        515                 520                 525

Lys His Val Arg Tyr Gln Met Pro Val Pro Ser Lys Ile Pro Arg Lys
    530                 535                 540

Ala Ser Ser Leu Thr Pro Leu Ala Ala Gln Lys Gln
545                 550                 555
```

<210> 141
<211> 687
<212> PRT
<213> Homo sapiens

<400> 141

```
Met Ala Glu Glu Leu Val Leu Glu Arg Cys Asp Leu Glu Leu Glu Thr
1               5                  10                  15

Asn Gly Arg Asp His His Thr Ala Asp Leu Cys Arg Glu Lys Leu Val
            20                  25                  30

Val Arg Arg Gly Gln Pro Phe Trp Leu Thr Leu His Phe Glu Gly Arg
        35                  40                  45

Asn Tyr Gln Ala Ser Val Asp Ser Leu Thr Phe Ser Val Val Thr Gly
    50                  55                  60

Pro Ala Pro Ser Gln Glu Ala Gly Thr Lys Ala Arg Phe Pro Leu Arg
65                  70                  75                  80

Asp Ala Val Glu Glu Gly Asp Trp Thr Ala Thr Val Val Asp Gln Gln
                85                  90                  95

Asp Cys Thr Leu Ser Leu Gln Leu Thr Thr Pro Ala Asn Ala Pro Ile
            100                 105                 110

Gly Leu Tyr Arg Leu Ser Leu Glu Ala Ser Thr Gly Tyr Gln Gly Ser
        115                 120                 125

Ser Phe Val Leu Gly His Phe Ile Leu Leu Phe Asn Ala Trp Cys Pro
    130                 135                 140

Ala Asp Ala Val Tyr Leu Asp Ser Glu Glu Glu Arg Gln Glu Tyr Val
145                 150                 155                 160

Leu Thr Gln Gln Gly Phe Ile Tyr Gln Gly Ser Ala Lys Phe Ile Lys
                165                 170                 175

Asn Ile Pro Trp Asn Phe Gly Gln Phe Gln Asp Gly Ile Leu Asp Ile
            180                 185                 190

Cys Leu Ile Leu Leu Asp Val Asn Pro Lys Phe Leu Lys Asn Ala Gly
        195                 200                 205

Arg Asp Cys Ser Arg Arg Ser Ser Pro Val Tyr Val Gly Arg Val Gly
    210                 215                 220

Ser Gly Met Val Asn Cys Asn Asp Asp Gln Gly Val Leu Leu Gly Arg
225                 230                 235                 240

Trp Asp Asn Asn Tyr Gly Asp Gly Val Ser Pro Met Ser Trp Ile Gly
```

245 250 255

Ser Val Asp Ile Leu Arg Arg Trp Lys Asn His Gly Cys Gln Arg Val
260 265 270

Lys Tyr Gly Gln Cys Trp Val Phe Ala Ala Val Ala Cys Thr Val Leu
275 280 285

Arg Cys Leu Gly Ile Pro Thr Arg Val Val Thr Asn Tyr Asn Ser Ala
290 295 300

His Asp Gln Asn Ser Asn Leu Leu Ile Glu Tyr Phe Arg Asn Glu Phe
305 310 315 320

Gly Glu Ile Gln Gly Asp Lys Ser Glu Met Ile Trp Asn Phe His Cys
325 330 335

Trp Val Glu Ser Trp Met Thr Arg Pro Asp Leu Gln Pro Gly Tyr Glu
340 345 350

Gly Trp Gln Ala Leu Asp Pro Thr Pro Gln Glu Lys Ser Glu Gly Thr
355 360 365

Tyr Cys Cys Gly Pro Val Pro Val Arg Ala Ile Lys Glu Gly Asp Leu
370 375 380

Ser Thr Lys Tyr Asp Ala Pro Phe Val Phe Ala Glu Val Asn Ala Asp
385 390 395 400

Val Val Asp Trp Ile Gln Gln Asp Asp Gly Ser Val His Lys Ser Ile
405 410 415

Asn Arg Ser Leu Ile Val Gly Leu Lys Ile Ser Thr Lys Ser Val Gly
420 425 430

Arg Asp Glu Arg Glu Asp Ile Thr His Thr Tyr Lys Tyr Pro Glu Gly
435 440 445

Ser Ser Glu Glu Arg Glu Ala Phe Thr Arg Ala Asn His Leu Asn Lys
450 455 460

Leu Ala Glu Lys Glu Glu Thr Gly Met Ala Met Arg Ile Arg Val Gly
465 470 475 480

Gln Ser Met Asn Met Gly Ser Asp Phe Asp Val Phe Ala His Ile Thr
485 490 495

Asn Asn Thr Ala Glu Glu Tyr Val Cys Arg Leu Leu Leu Cys Ala Arg
500 505 510

Thr Val Ser Tyr Asn Gly Ile Leu Gly Pro Glu Cys Gly Thr Lys Tyr

515 520 525

Leu Leu Asn Leu Thr Leu Glu Pro Phe Ser Glu Lys Ser Val Pro Leu
530 535 540

Cys Ile Leu Tyr Glu Lys Tyr Arg Asp Cys Leu Thr Glu Ser Asn Leu
545 550 555 560

Ile Lys Val Arg Ala Leu Leu Val Glu Pro Val Ile Asn Ser Tyr Leu
565 570 575

Leu Ala Glu Arg Asp Leu Tyr Leu Glu Asn Pro Glu Ile Lys Ile Arg
580 585 590

Ile Leu Gly Glu Pro Lys Gln Lys Arg Lys Leu Val Ala Glu Val Ser
595 600 605

Leu Gln Asn Pro Leu Pro Val Ala Leu Glu Gly Cys Thr Phe Thr Val
610 615 620

Glu Gly Ala Gly Leu Thr Glu Glu Gln Lys Thr Val Glu Ile Pro Asp
625 630 635 640

Pro Val Glu Ala Gly Glu Glu Val Lys Val Arg Met Asp Leu Val Pro
645 650 655

Leu His Met Gly Leu His Lys Leu Val Val Asn Phe Glu Ser Asp Lys
660 665 670

Leu Lys Ala Val Lys Gly Phe Arg Asn Val Ile Ile Gly Pro Ala
675 680 685

<210> 142
<211> 164
<212> PRT
<213> Homo sapiens

<400> 142

Met Ser Val Asp Pro Leu Ser Ser Lys Ala Leu Lys Ile Lys Arg Glu
1 5 10 15

Leu Ser Glu Asn Thr Pro His Leu Ser Asp Glu Ala Leu Met Gly Leu
20 25 30

Ser Val Arg Glu Leu Asn Arg His Leu Arg Gly Leu Ser Ala Glu Glu
35 40 45

Val Thr Arg Leu Lys Gln Arg Arg Arg Thr Leu Lys Asn Arg Gly Tyr
50 55 60

Ala Ala Ser Cys Arg Val Lys Arg Val Cys Gln Lys Glu Glu Leu Gln
65 70 75 80

```
Lys Gln Lys Ser Glu Leu Glu Arg Glu Val Asp Lys Leu Ala Arg Glu
                85                  90                  95

Asn Ala Ala Met Arg Leu Glu Leu Asp Ala Leu Arg Gly Lys Cys Glu
            100                 105                 110

Ala Leu Gln Gly Phe Ala Arg Ser Val Ala Ala Ala Arg Gly Pro Ala
        115                 120                 125

Thr Leu Val Ala Pro Ala Ser Val Ile Thr Ile Val Lys Ser Thr Pro
    130                 135                 140

Gly Ser Gly Ser Gly Pro Ala His Gly Pro Asp Pro Ala His Gly Pro
145                 150                 155                 160

Ala Ser Cys Ser
```

<210> 143
<211> 433
<212> PRT
<213> Homo sapiens

<400> 143

```
Met Glu Arg Met Leu Pro Leu Leu Ala Leu Gly Leu Leu Ala Ala Gly
1               5                   10                  15

Phe Cys Pro Ala Val Leu Cys His Pro Asn Ser Pro Leu Asp Glu Glu
            20                  25                  30

Asn Leu Thr Gln Glu Asn Gln Asp Arg Gly Thr His Val Asp Leu Gly
        35                  40                  45

Leu Ala Ser Ala Asn Val Asp Phe Ala Phe Ser Leu Tyr Lys Gln Leu
    50                  55                  60

Val Leu Lys Ala Leu Asp Lys Asn Val Ile Phe Ser Pro Leu Ser Ile
65                  70                  75                  80

Ser Thr Ala Leu Ala Phe Leu Ser Leu Gly Ala His Asn Thr Thr Leu
                85                  90                  95

Thr Glu Ile Leu Lys Ala Ser Ser Ser Pro His Gly Asp Leu Leu Arg
            100                 105                 110

Gln Lys Phe Thr Gln Ser Phe Gln His Leu Arg Ala Pro Ser Ile Ser
        115                 120                 125

Ser Ser Asp Glu Leu Gln Leu Ser Met Gly Asn Ala Met Phe Val Lys
    130                 135                 140
```

```
Glu Gln Leu Ser Leu Leu Asp Arg Phe Thr Glu Asp Ala Lys Arg Leu
145                 150                 155                 160

Tyr Gly Ser Glu Ala Phe Ala Thr Asp Phe Gln Asp Ser Ala Ala Ala
                165                 170                 175

Lys Lys Leu Ile Asn Asp Tyr Val Lys Asn Gly Thr Arg Gly Lys Ile
            180                 185                 190

Thr Asp Leu Ile Lys Asp Pro Asp Ser Gln Thr Met Met Val Leu Val
        195                 200                 205

Asn Tyr Ile Phe Phe Lys Ala Lys Trp Glu Met Pro Phe Asp Pro Gln
    210                 215                 220

Asp Thr His Gln Ser Arg Phe Tyr Leu Ser Lys Lys Lys Trp Val Met
225                 230                 235                 240

Val Pro Met Met Ser Leu His His Leu Thr Ile Pro Tyr Phe Arg Asp
                245                 250                 255

Glu Glu Leu Ser Cys Thr Val Val Glu Leu Lys Tyr Thr Gly Asn Ala
            260                 265                 270

Ser Ala Leu Phe Ile Leu Pro Asp Gln Asp Lys Met Glu Glu Val Glu
        275                 280                 285

Ala Met Leu Leu Pro Glu Thr Leu Lys Arg Trp Arg Asp Ser Leu Glu
    290                 295                 300

Phe Arg Glu Ile Gly Glu Leu Tyr Leu Pro Lys Phe Ser Ile Ser Arg
305                 310                 315                 320

Asp Tyr Asn Leu Asn Asp Ile Leu Leu Gln Leu Gly Ile Glu Glu Ala
                325                 330                 335

Phe Thr Ser Lys Ala Asp Leu Ser Gly Ile Thr Gly Ala Arg Asn Leu
            340                 345                 350

Ala Val Ser Gln Val Val His Lys Val Val Ser Asp Val Phe Glu Glu
        355                 360                 365

Gly Thr Glu Ala Ser Ala Ala Thr Ala Val Lys Ile Thr Leu Leu Ser
    370                 375                 380

Ala Leu Val Glu Thr Arg Thr Ile Val Arg Phe Asn Arg Pro Phe Leu
385                 390                 395                 400

Met Ile Ile Val Pro Thr Asp Thr Gln Asn Ile Phe Phe Met Ser Lys
                405                 410                 415
```

Val Thr Asn Pro Ser Lys Pro Arg Ala Cys Ile Lys Gln Trp Gly Ser
420 425 430

Gln

<210> 144
<211> 107
<212> PRT
<213> Homo sapiens

<400> 144

Met Ala Arg Ala Ala Leu Ser Ala Ala Pro Ser Asn Pro Arg Leu Leu
1 5 10 15

Arg Val Ala Leu Leu Leu Leu Leu Leu Val Ala Ala Gly Arg Arg Ala
20 25 30

Ala Gly Ala Ser Val Ala Thr Glu Leu Arg Cys Gln Cys Leu Gln Thr
35 40 45

Leu Gln Gly Ile His Pro Lys Asn Ile Gln Ser Val Asn Val Lys Ser
50 55 60

Pro Gly Pro His Cys Ala Gln Thr Glu Val Ile Ala Thr Leu Lys Asn
65 70 75 80

Gly Arg Lys Ala Cys Leu Asn Pro Ala Ser Pro Ile Val Lys Lys Ile
85 90 95

Ile Glu Lys Met Leu Asn Ser Asp Lys Ser Asn
100 105

<210> 145
<211> 375
<212> PRT
<213> Homo sapiens

<400> 145

Met Glu Arg Ala Ser Cys Leu Leu Leu Leu Leu Leu Pro Leu Val His
1 5 10 15

Val Ser Ala Thr Thr Pro Glu Pro Cys Glu Leu Asp Asp Glu Asp Phe
20 25 30

Arg Cys Val Cys Asn Phe Ser Glu Pro Gln Pro Asp Trp Ser Glu Ala
35 40 45

Phe Gln Cys Val Ser Ala Val Glu Val Glu Ile His Ala Gly Gly Leu
50 55 60

Asn Leu Glu Pro Phe Leu Lys Arg Val Asp Ala Asp Ala Asp Pro Arg

65 70 75 80

Gln Tyr Ala Asp Thr Val Lys Ala Leu Arg Val Arg Arg Leu Thr Val
85 90 95

Gly Ala Ala Gln Val Pro Ala Gln Leu Leu Val Gly Ala Leu Arg Val
100 105 110

Leu Ala Tyr Ser Arg Leu Lys Glu Leu Thr Leu Glu Asp Leu Lys Ile
115 120 125

Thr Gly Thr Met Pro Pro Leu Pro Leu Glu Ala Thr Gly Leu Ala Leu
130 135 140

Ser Ser Leu Arg Leu Arg Asn Val Ser Trp Ala Thr Gly Arg Ser Trp
145 150 155 160

Leu Ala Glu Leu Gln Gln Trp Leu Lys Pro Gly Leu Lys Val Leu Ser
165 170 175

Ile Ala Gln Ala His Ser Pro Ala Phe Ser Cys Glu Gln Val Arg Ala
180 185 190

Phe Pro Ala Leu Thr Ser Leu Asp Leu Ser Asp Asn Pro Gly Leu Gly
195 200 205

Glu Arg Gly Leu Met Ala Ala Leu Cys Pro His Lys Phe Pro Ala Ile
210 215 220

Gln Asn Leu Ala Leu Arg Asn Thr Gly Met Glu Thr Pro Thr Gly Val
225 230 235 240

Cys Ala Ala Leu Ala Ala Ala Gly Val Gln Pro His Ser Leu Asp Leu
245 250 255

Ser His Asn Ser Leu Arg Ala Thr Val Asn Pro Ser Ala Pro Arg Cys
260 265 270

Met Trp Ser Ser Ala Leu Asn Ser Leu Asn Leu Ser Phe Ala Gly Leu
275 280 285

Glu Gln Val Pro Lys Gly Leu Pro Ala Lys Leu Arg Val Leu Asp Leu
290 295 300

Ser Cys Asn Arg Leu Asn Arg Ala Pro Gln Pro Asp Glu Leu Pro Glu
305 310 315 320

Val Asp Asn Leu Thr Leu Asp Gly Asn Pro Phe Leu Val Pro Gly Thr
325 330 335

Ala Leu Pro His Glu Gly Ser Met Asn Ser Gly Val Val Pro Ala Cys

340 345 350

Ala Arg Ser Thr Leu Ser Val Gly Val Ser Gly Thr Leu Val Leu Leu
355 360 365

Gln Gly Ala Arg Gly Phe Ala
370 375

<210> 146
<211> 1419
<212> PRT
<213> Homo sapiens

<400> 146

Met Asp Gly Gly Gly Val Cys Val Gly Arg Gly Asp Leu Leu Ser Ser
1 5 10 15

Pro Gln Ala Leu Gly Gln Leu Leu Arg Lys Glu Ser Arg Pro Arg Arg
20 25 30

Ala Met Lys Pro Arg Lys Ala Glu Pro His Ser Phe Arg Glu Lys Val
35 40 45

Phe Arg Lys Lys Pro Pro Val Cys Ala Val Cys Lys Val Thr Ile Asp
50 55 60

Gly Thr Gly Val Ser Cys Arg Val Cys Lys Val Ala Thr His Arg Lys
65 70 75 80

Cys Glu Ala Lys Val Thr Ser Ala Cys Gln Ala Leu Pro Pro Val Glu
85 90 95

Leu Arg Arg Asn Thr Ala Pro Val Arg Arg Ile Glu His Leu Gly Ser
100 105 110

Thr Lys Ser Leu Asn His Ser Lys Gln Arg Ser Thr Leu Pro Arg Ser
115 120 125

Phe Ser Leu Asp Pro Leu Met Glu Arg Arg Trp Asp Leu Asp Leu Thr
130 135 140

Tyr Val Thr Glu Arg Ile Leu Ala Ala Ala Phe Pro Ala Arg Pro Asp
145 150 155 160

Glu Gln Arg His Arg Gly His Leu Arg Glu Leu Ala His Val Leu Gln
165 170 175

Ser Lys His Arg Asp Lys Tyr Leu Leu Phe Asn Leu Ser Glu Lys Arg
180 185 190

His Asp Leu Thr Arg Leu Asn Pro Lys Val Gln Asp Phe Gly Trp Pro
195 200 205

```
Glu Leu His Ala Pro Pro Leu Asp Lys Leu Cys Ser Ile Cys Lys Ala
    210                 215                 220

Met Glu Thr Trp Leu Ser Ala Asp Pro Gln His Val Val Val Leu Tyr
225                 230                 235                 240

Cys Lys Gly Asn Lys Gly Lys Leu Gly Val Ile Val Ser Ala Tyr Met
                245                 250                 255

His Tyr Ser Lys Ile Ser Ala Gly Ala Asp Gln Ala Leu Ala Thr Leu
            260                 265                 270

Thr Met Arg Lys Phe Cys Glu Asp Lys Val Ala Thr Glu Leu Gln Pro
        275                 280                 285

Ser Gln Arg Arg Tyr Ile Ser Tyr Phe Ser Gly Leu Leu Ser Gly Ser
    290                 295                 300

Ile Arg Met Asn Ser Ser Pro Leu Phe Leu His Tyr Val Leu Ile Pro
305                 310                 315                 320

Met Leu Pro Ala Phe Glu Pro Gly Thr Gly Phe Gln Pro Phe Leu Lys
                325                 330                 335

Ile Tyr Gln Ser Met Gln Leu Val Tyr Thr Ser Gly Val Tyr His Ile
            340                 345                 350

Ala Gly Pro Gly Pro Gln Gln Leu Cys Ile Ser Leu Glu Pro Ala Leu
        355                 360                 365

Leu Leu Lys Gly Asp Val Met Val Thr Cys Tyr His Lys Gly Gly Arg
    370                 375                 380

Gly Thr Asp Arg Thr Leu Val Phe Arg Val Gln Phe His Thr Cys Thr
385                 390                 395                 400

Ile His Gly Pro Gln Leu Thr Phe Pro Lys Asp Gln Leu Asp Glu Ala
                405                 410                 415

Trp Thr Asp Glu Arg Phe Pro Phe Gln Ala Ser Val Glu Phe Val Phe
            420                 425                 430

Ser Ser Ser Pro Glu Lys Ile Lys Gly Ser Thr Pro Arg Asn Asp Pro
        435                 440                 445

Ser Val Ser Val Asp Tyr Asn Thr Thr Glu Pro Ala Val Arg Trp Asp
    450                 455                 460

Ser Tyr Glu Asn Phe Asn Gln His His Glu Asp Ser Val Asp Gly Ser
465                 470                 475                 480
```

```
Leu Thr His Thr Arg Gly Pro Leu Asp Gly Ser Pro Tyr Ala Gln Val
                485                 490                 495

Gln Arg Pro Pro Arg Gln Thr Pro Pro Ala Pro Ser Pro Glu Pro Pro
            500                 505                 510

Pro Pro Pro Met Leu Ser Val Ser Ser Asp Ser Gly His Ser Ser Thr
        515                 520                 525

Leu Thr Thr Glu Pro Ala Ala Glu Ser Pro Gly Arg Pro Pro Pro Thr
    530                 535                 540

Ala Ala Glu Arg Gln Glu Leu Asp Arg Leu Leu Gly Gly Cys Gly Val
545                 550                 555                 560

Ala Ser Gly Gly Arg Gly Ala Gly Arg Glu Thr Ala Ile Leu Asp Asp
                565                 570                 575

Glu Glu Gln Pro Thr Val Gly Gly Gly Pro His Leu Gly Val Tyr Pro
            580                 585                 590

Gly His Arg Pro Gly Leu Ser Arg His Cys Ser Cys Arg Gln Gly Tyr
        595                 600                 605

Arg Glu Pro Cys Gly Val Pro Asn Gly Gly Tyr Tyr Arg Pro Glu Gly
    610                 615                 620

Thr Leu Glu Arg Arg Arg Leu Ala Tyr Gly Gly Tyr Glu Gly Ser Pro
625                 630                 635                 640

Gln Gly Tyr Ala Glu Ala Ser Met Glu Lys Arg Arg Leu Cys Arg Ser
                645                 650                 655

Leu Ser Glu Gly Leu Tyr Pro Tyr Pro Pro Glu Met Gly Lys Pro Ala
            660                 665                 670

Thr Gly Asp Phe Gly Tyr Arg Ala Pro Gly Tyr Arg Glu Val Val Ile
        675                 680                 685

Leu Glu Asp Pro Gly Leu Pro Ala Leu Tyr Pro Cys Pro Ala Cys Glu
    690                 695                 700

Glu Lys Leu Ala Leu Pro Thr Ala Ala Leu Tyr Gly Leu Arg Leu Glu
705                 710                 715                 720

Arg Glu Ala Gly Glu Gly Trp Ala Ser Glu Ala Gly Lys Pro Leu Leu
                725                 730                 735

His Pro Val Arg Pro Gly His Pro Leu Pro Leu Leu Leu Pro Ala Cys
            740                 745                 750
```

```
Gly His His His Ala Pro Met Pro Asp Tyr Ser Cys Leu Lys Pro Pro
        755                 760                 765

Lys Ala Gly Glu Glu Gly His Glu Gly Cys Ser Tyr Thr Met Cys Pro
    770                 775                 780

Glu Gly Arg Tyr Gly His Pro Gly Tyr Pro Ala Leu Val Thr Tyr Ser
785                 790                 795                 800

Tyr Gly Gly Ala Val Pro Ser Tyr Cys Pro Ala Tyr Gly Arg Val Pro
                805                 810                 815

His Ser Cys Gly Ser Pro Gly Glu Gly Arg Gly Tyr Pro Ser Pro Gly
            820                 825                 830

Ala His Ser Pro Arg Ala Gly Ser Ile Ser Pro Gly Ser Pro Pro Tyr
        835                 840                 845

Pro Gln Ser Arg Lys Leu Ser Tyr Glu Ile Pro Thr Glu Glu Gly Gly
    850                 855                 860

Asp Arg Tyr Pro Leu Pro Gly His Leu Ala Ser Ala Gly Pro Leu Ala
865                 870                 875                 880

Ser Ala Glu Ser Leu Glu Pro Val Ser Trp Arg Glu Gly Pro Ser Gly
                885                 890                 895

His Ser Thr Leu Pro Arg Ser Pro Arg Asp Ala Pro Cys Ser Ala Ser
            900                 905                 910

Ser Glu Leu Ser Gly Pro Ser Thr Pro Leu His Thr Ser Ser Pro Val
        915                 920                 925

Gln Gly Lys Glu Ser Thr Arg Arg Gln Asp Thr Arg Ser Pro Thr Ser
    930                 935                 940

Ala Pro Thr Gln Arg Leu Ser Pro Gly Glu Ala Leu Pro Pro Val Ser
945                 950                 955                 960

Gln Ala Gly Thr Gly Lys Ala Pro Glu Leu Pro Ser Gly Ser Gly Pro
                965                 970                 975

Glu Pro Leu Ala Pro Ser Pro Val Ser Pro Thr Phe Pro Pro Ser Ser
            980                 985                 990

Pro Ser Asp Trp Pro Gln Glu Arg  Ser Pro Gly Gly His  Ser Asp Gly
        995                 1000                1005

Ala Ser  Pro Arg Ser Pro Val  Pro Thr Thr Leu Pro  Gly Leu Arg
    1010                1015                1020
```

```
His Ala  Pro Trp Gln Gly Pro  Arg Gly Pro Pro Asp  Ser Pro Asp
    1025                 1030                 1035

Gly Ser  Pro Leu Thr Pro Val  Pro Ser Gln Met Pro  Trp Leu Val
    1040                 1045                 1050

Ala Ser  Pro Glu Pro Pro Gln  Ser Ser Pro Thr Pro  Ala Phe Pro
    1055                 1060                 1065

Leu Ala  Ala Ser Tyr Asp Thr  Asn Gly Leu Ser Gln  Pro Pro Leu
    1070                 1075                 1080

Pro Glu  Lys Arg His Leu Pro  Gly Pro Gly Gln Gln  Pro Gly Pro
    1085                 1090                 1095

Trp Gly  Pro Glu Gln Ala Ser  Ser Pro Ala Arg Gly  Ile Ser His
    1100                 1105                 1110

His Val  Thr Phe Ala Pro Leu  Leu Ser Asp Asn Val  Pro Gln Thr
    1115                 1120                 1125

Pro Glu  Pro Pro Thr Gln Glu  Ser Gln Ser Asn Val  Lys Phe Val
    1130                 1135                 1140

Gln Asp  Thr Ser Lys Phe Trp  Tyr Lys Pro His Leu  Ser Arg Asp
    1145                 1150                 1155

Gln Ala  Ile Ala Leu Leu Lys  Asp Lys Asp Pro Gly  Ala Phe Leu
    1160                 1165                 1170

Ile Arg  Asp Ser His Ser Phe  Gln Gly Ala Tyr Gly  Leu Ala Leu
    1175                 1180                 1185

Lys Val  Ala Thr Pro Pro Pro  Ser Ala Gln Pro Trp  Lys Gly Asp
    1190                 1195                 1200

Pro Val  Glu Gln Leu Val Arg  His Phe Leu Ile Glu  Thr Gly Pro
    1205                 1210                 1215

Lys Gly  Val Lys Ile Lys Gly  Cys Pro Ser Glu Pro  Tyr Phe Gly
    1220                 1225                 1230

Ser Leu  Ser Ala Leu Val Ser  Gln His Ser Ile Ser  Pro Ile Ser
    1235                 1240                 1245

Leu Pro  Cys Cys Leu Arg Ile  Leu Ser Lys Asp Pro  Leu Glu Glu
    1250                 1255                 1260

Thr Pro  Glu Ala Pro Val Pro  Thr Asn Met Ser Thr  Ala Ala Asp
    1265                 1270                 1275
```

```
Leu Leu  Arg Gln Gly Ala Ala  Cys Ser Val Leu Tyr  Leu Thr Ser
    1280                 1285                 1290

Val Glu  Thr Glu Ser Leu Thr  Gly Pro Gln Ala Val  Ala Arg Ala
    1295                 1300                 1305

Ser Ser  Ala Ala Leu Ser Cys  Ser Pro Arg Pro Thr  Pro Ala Val
    1310                 1315                 1320

Val His  Phe Lys Val Ser Ala  Gln Gly Ile Thr Leu  Thr Asp Asn
    1325                 1330                 1335

Gln Arg  Lys Leu Phe Phe Arg  Arg His Tyr Pro Val  Asn Ser Ile
    1340                 1345                 1350

Thr Phe  Ser Ser Thr Asp Pro  Gln Asp Arg Arg Trp  Thr Asn Pro
    1355                 1360                 1365

Asp Gly  Thr Thr Ser Lys Ile  Phe Gly Phe Val Ala  Lys Lys Pro
    1370                 1375                 1380

Gly Ser  Pro Trp Glu Asn Val  Cys His Leu Phe Ala  Glu Leu Asp
    1385                 1390                 1395

Pro Asp  Gln Pro Ala Gly Ala  Ile Val Thr Phe Ile  Thr Lys Val
    1400                 1405                 1410

Leu Leu  Gly Gln Arg Lys
    1415
```

<210> 147
<211> 383
<212> PRT
<213> Homo sapiens

<400> 147

```
Met Gly Val Lys Ala Ser Gln Thr Gly Phe Val Val Leu Val Leu Leu
1               5                  10                  15

Gln Cys Cys Ser Ala Tyr Lys Leu Val Cys Tyr Tyr Thr Ser Trp Ser
            20                  25                  30

Gln Tyr Arg Glu Gly Asp Gly Ser Cys Phe Pro Asp Ala Leu Asp Arg
        35                  40                  45

Phe Leu Cys Thr His Ile Ile Tyr Ser Phe Ala Asn Ile Ser Asn Asp
    50                  55                  60

His Ile Asp Thr Trp Glu Trp Asn Asp Val Thr Leu Tyr Gly Met Leu
65                  70                  75                  80
```

```
Asn Thr Leu Lys Asn Arg Asn Pro Asn Leu Lys Thr Leu Leu Ser Val
                85                  90                  95

Gly Gly Trp Asn Phe Gly Ser Gln Arg Phe Ser Lys Ile Ala Ser Asn
            100                 105                 110

Thr Gln Ser Arg Arg Thr Phe Ile Lys Ser Val Pro Pro Phe Leu Arg
        115                 120                 125

Thr His Gly Phe Asp Gly Leu Asp Leu Ala Trp Leu Tyr Pro Gly Arg
    130                 135                 140

Arg Asp Lys Gln His Phe Thr Thr Leu Ile Lys Glu Met Lys Ala Glu
145                 150                 155                 160

Phe Ile Lys Glu Ala Gln Pro Gly Lys Lys Gln Leu Leu Leu Ser Ala
                165                 170                 175

Ala Leu Ser Ala Gly Lys Val Thr Ile Asp Ser Ser Tyr Asp Ile Ala
            180                 185                 190

Lys Ile Ser Gln His Leu Asp Phe Ile Ser Ile Met Thr Tyr Asp Phe
        195                 200                 205

His Gly Ala Trp Arg Gly Thr Thr Gly His His Ser Pro Leu Phe Arg
    210                 215                 220

Gly Gln Glu Asp Ala Ser Pro Asp Arg Phe Ser Asn Thr Asp Tyr Ala
225                 230                 235                 240

Val Gly Tyr Met Leu Arg Leu Gly Ala Pro Ala Ser Lys Leu Val Met
                245                 250                 255

Gly Ile Pro Thr Phe Gly Arg Ser Phe Thr Leu Ala Ser Ser Glu Thr
            260                 265                 270

Gly Val Gly Ala Pro Ile Ser Gly Pro Gly Ile Pro Gly Arg Phe Thr
        275                 280                 285

Lys Glu Ala Gly Thr Leu Ala Tyr Tyr Glu Ile Cys Asp Phe Leu Arg
    290                 295                 300

Gly Ala Thr Val His Arg Thr Leu Gly Gln Gln Val Pro Tyr Ala Thr
305                 310                 315                 320

Lys Gly Asn Gln Trp Val Gly Tyr Asp Asp Gln Glu Ser Val Lys Ser
                325                 330                 335

Lys Val Gln Tyr Leu Lys Asp Arg Gln Leu Ala Gly Ala Met Val Trp
            340                 345                 350
```

```
Ala Leu Asp Leu Asp Asp Phe Gln Gly Ser Phe Cys Gly Gln Asp Leu
        355                 360                 365

Arg Phe Pro Leu Thr Asn Ala Ile Lys Asp Ala Leu Ala Ala Thr
    370                 375                 380
```

<210> 148
<211> 417
<212> PRT
<213> Homo sapiens

<400> 148

```
Met Arg Ser Leu Leu Leu Gly Thr Leu Cys Leu Leu Ala Val Ala Leu
1               5                  10                  15

Ala Ala Glu Val Lys Lys Pro Val Glu Ala Ala Ala Pro Gly Thr Ala
            20                  25                  30

Glu Lys Leu Ser Ser Lys Ala Thr Thr Leu Ala Glu Pro Ser Thr Gly
        35                  40                  45

Leu Ala Phe Ser Leu Tyr Gln Ala Met Ala Lys Asp Gln Ala Val Glu
    50                  55                  60

Asn Ile Leu Val Ser Pro Val Val Val Ala Ser Ser Leu Gly Leu Val
65                  70                  75                  80

Ser Leu Gly Gly Lys Ala Thr Thr Ala Ser Gln Ala Lys Ala Val Leu
                85                  90                  95

Ser Ala Glu Gln Leu Arg Asp Glu Glu Val His Ala Gly Leu Gly Glu
            100                 105                 110

Leu Leu Arg Ser Leu Ser Asn Ser Thr Ala Arg Asn Val Thr Trp Lys
        115                 120                 125

Leu Gly Ser Arg Leu Tyr Gly Pro Ser Ser Val Ser Phe Ala Asp Asp
    130                 135                 140

Phe Val Arg Ser Ser Lys Gln His Tyr Asn Cys Glu His Ser Lys Ile
145                 150                 155                 160

Asn Phe Pro Asp Lys Arg Ser Ala Leu Gln Ser Ile Asn Glu Trp Ala
                165                 170                 175

Ala Gln Thr Thr Asp Gly Lys Leu Pro Glu Val Thr Lys Asp Val Glu
            180                 185                 190

Arg Thr Asp Gly Ala Leu Leu Val Asn Ala Met Phe Phe Lys Pro His
        195                 200                 205
```

```
Trp Asp Glu Lys Phe His His Lys Met Val Asp Asn Arg Gly Phe Met
    210                 215                 220

Val Thr Arg Ser Tyr Thr Val Gly Val Thr Met Met His Arg Thr Gly
225                 230                 235                 240

Leu Tyr Asn Tyr Tyr Asp Asp Glu Lys Glu Lys Leu Gln Leu Val Glu
                245                 250                 255

Met Pro Leu Ala His Lys Leu Ser Ser Leu Ile Ile Leu Met Pro His
            260                 265                 270

His Val Glu Pro Leu Glu Arg Leu Glu Lys Leu Leu Thr Lys Glu Gln
        275                 280                 285

Leu Lys Ile Trp Met Gly Lys Met Gln Lys Lys Ala Val Ala Ile Ser
    290                 295                 300

Leu Pro Lys Gly Val Val Glu Val Thr His Asp Leu Gln Lys His Leu
305                 310                 315                 320

Ala Gly Leu Gly Leu Thr Glu Ala Ile Asp Lys Asn Lys Ala Asp Leu
                325                 330                 335

Ser Arg Met Ser Gly Lys Lys Asp Leu Tyr Leu Ala Ser Val Phe His
            340                 345                 350

Ala Thr Ala Phe Glu Leu Asp Thr Asp Gly Asn Pro Phe Asp Gln Asp
        355                 360                 365

Ile Tyr Gly Arg Glu Glu Leu Arg Ser Pro Lys Leu Phe Tyr Ala Asp
    370                 375                 380

His Pro Phe Ile Phe Leu Val Arg Asp Thr Gln Ser Gly Ser Leu Leu
385                 390                 395                 400

Phe Ile Gly Arg Leu Val Arg Leu Lys Gly Asp Lys Met Arg Asp Glu
                405                 410                 415

Leu
```

<210> 149
<211> 61
<212> PRT
<213> Homo sapiens

<400> 149

```
Met Asp Pro Asn Cys Ser Cys Ser Pro Val Gly Ser Cys Ala Cys Ala
1               5                   10                  15

Gly Ser Cys Lys Cys Lys Glu Cys Lys Cys Thr Ser Cys Lys Lys Ser
```

20 25 30

Cys Cys Ser Cys Cys Pro Val Gly Cys Ala Lys Cys Ala Gln Gly Cys
35 40 45

Ile Cys Lys Gly Thr Ser Asp Lys Cys Ser Cys Cys Ala
50 55 60

<210> 150
<211> 1651
<212> PRT
<213> Homo sapiens

<400> 150

Met Ala Pro Arg Ala Ala Gly Gly Ala Pro Leu Ser Ala Arg Ala Ala
1 5 10 15

Ala Ala Ser Pro Pro Pro Phe Gln Thr Pro Pro Arg Cys Pro Val Pro
20 25 30

Leu Leu Leu Leu Leu Leu Leu Gly Ala Ala Arg Ala Gly Ala Leu Glu
35 40 45

Ile Gln Arg Arg Phe Pro Ser Pro Thr Pro Thr Asn Asn Phe Ala Leu
50 55 60

Asp Gly Ala Ala Gly Thr Val Tyr Leu Ala Ala Val Asn Arg Leu Tyr
65 70 75 80

Gln Leu Ser Gly Ala Asn Leu Ser Leu Glu Ala Glu Ala Ala Val Gly
85 90 95

Pro Val Pro Asp Ser Pro Leu Cys His Ala Pro Gln Leu Pro Gln Ala
100 105 110

Ser Cys Glu His Pro Arg Arg Leu Thr Asp Asn Tyr Asn Lys Ile Leu
115 120 125

Gln Leu Asp Pro Gly Gln Gly Leu Val Val Val Cys Gly Ser Ile Tyr
130 135 140

Gln Gly Phe Cys Gln Leu Arg Arg Arg Gly Asn Ile Ser Ala Val Ala
145 150 155 160

Val Arg Phe Pro Pro Ala Ala Pro Pro Ala Glu Pro Val Thr Val Phe
165 170 175

Pro Ser Met Leu Asn Val Ala Ala Asn His Pro Asn Ala Ser Thr Val
180 185 190

Gly Leu Val Leu Pro Pro Ala Ala Gly Ala Gly Gly Ser Arg Leu Leu
195 200 205

```
Val Gly Ala Thr Tyr Thr Gly Tyr Gly Ser Ser Phe Phe Pro Arg Asn
    210                 215                 220

Arg Ser Leu Glu Asp His Arg Phe Glu Asn Thr Pro Glu Ile Ala Ile
225                 230                 235                 240

Arg Ser Leu Asp Thr Arg Gly Asp Leu Ala Lys Leu Phe Thr Phe Asp
                245                 250                 255

Leu Asn Pro Ser Asp Asp Asn Ile Leu Lys Ile Lys Gln Gly Ala Lys
            260                 265                 270

Glu Gln His Lys Leu Gly Phe Val Ser Ala Phe Leu His Pro Ser Asp
        275                 280                 285

Pro Pro Pro Gly Ala Gln Ser Tyr Ala Tyr Leu Ala Leu Asn Ser Glu
    290                 295                 300

Ala Arg Ala Gly Asp Lys Glu Ser Gln Ala Arg Ser Leu Leu Ala Arg
305                 310                 315                 320

Ile Cys Leu Pro His Gly Ala Gly Gly Asp Ala Lys Lys Leu Thr Glu
                325                 330                 335

Ser Tyr Ile Gln Leu Gly Leu Gln Cys Ala Gly Gly Ala Gly Arg Gly
            340                 345                 350

Asp Leu Tyr Ser Arg Leu Val Ser Val Phe Pro Ala Arg Glu Arg Leu
        355                 360                 365

Phe Ala Val Phe Glu Arg Pro Gln Gly Ser Pro Ala Ala Arg Ala Ala
    370                 375                 380

Pro Ala Ala Leu Cys Ala Phe Arg Phe Ala Asp Val Arg Ala Ala Ile
385                 390                 395                 400

Arg Ala Ala Arg Thr Ala Cys Phe Val Glu Pro Ala Pro Asp Val Val
                405                 410                 415

Ala Val Leu Asp Ser Val Val Gln Gly Thr Gly Pro Ala Cys Glu Arg
            420                 425                 430

Lys Leu Asn Ile Gln Leu Gln Pro Glu Gln Leu Asp Cys Gly Ala Ala
        435                 440                 445

His Leu Gln His Pro Leu Ser Ile Leu Gln Pro Leu Lys Ala Thr Pro
    450                 455                 460

Val Phe Arg Ala Pro Gly Leu Thr Ser Val Ala Val Ala Ser Val Asn
465                 470                 475                 480
```

```
Asn Tyr Thr Ala Val Phe Leu Gly Thr Val Asn Gly Arg Leu Leu Lys
                485                 490                 495

Ile Asn Leu Asn Glu Ser Met Gln Val Val Ser Arg Arg Val Val Thr
            500                 505                 510

Val Ala Tyr Gly Glu Pro Val His His Val Met Gln Phe Asp Pro Ala
        515                 520                 525

Asp Ser Gly Tyr Leu Tyr Leu Met Thr Ser His Gln Met Ala Arg Val
    530                 535                 540

Lys Val Ala Ala Cys Asn Val His Ser Thr Cys Gly Asp Cys Val Gly
545                 550                 555                 560

Ala Ala Asp Ala Tyr Cys Gly Trp Cys Ala Leu Glu Thr Arg Cys Thr
                565                 570                 575

Leu Gln Gln Asp Cys Thr Asn Ser Ser Gln Gln His Phe Trp Thr Ser
            580                 585                 590

Ala Ser Glu Gly Pro Ser Arg Cys Pro Ala Met Thr Val Leu Pro Ser
        595                 600                 605

Glu Ile Asp Val Arg Gln Glu Tyr Pro Gly Met Ile Leu Gln Ile Ser
    610                 615                 620

Gly Ser Leu Pro Ser Leu Ser Gly Met Glu Met Ala Cys Asp Tyr Gly
625                 630                 635                 640

Asn Asn Ile Arg Thr Val Ala Arg Val Pro Gly Pro Ala Phe Gly His
                645                 650                 655

Gln Ile Ala Tyr Cys Asn Leu Leu Pro Arg Asp Gln Phe Pro Pro Phe
            660                 665                 670

Pro Pro Asn Gln Asp His Val Thr Val Glu Met Ser Val Arg Val Asn
        675                 680                 685

Gly Arg Asn Ile Val Lys Ala Asn Phe Thr Ile Tyr Asp Cys Ser Arg
    690                 695                 700

Thr Ala Gln Val Tyr Pro His Thr Ala Cys Thr Ser Cys Leu Ser Ala
705                 710                 715                 720

Gln Trp Pro Cys Phe Trp Cys Ser Gln Gln His Ser Cys Val Ser Asn
                725                 730                 735

Gln Ser Arg Cys Glu Ala Ser Pro Asn Pro Thr Ser Pro Gln Asp Cys
            740                 745                 750
```

```
Pro Arg Thr Leu Leu Ser Pro Leu Ala Pro Val Pro Thr Gly Gly Ser
        755                 760                 765

Gln Asn Ile Leu Val Pro Leu Ala Asn Thr Ala Phe Phe Gln Gly Ala
    770                 775                 780

Ala Leu Glu Cys Ser Phe Gly Leu Glu Glu Ile Phe Glu Ala Val Trp
785                 790                 795                 800

Val Asn Glu Ser Val Val Arg Cys Asp Gln Val Val Leu His Thr Thr
                805                 810                 815

Arg Lys Ser Gln Val Phe Pro Leu Ser Leu Gln Leu Lys Gly Arg Pro
            820                 825                 830

Ala Arg Phe Leu Asp Ser Pro Glu Pro Met Thr Val Met Val Tyr Asn
        835                 840                 845

Cys Ala Met Gly Ser Pro Asp Cys Ser Gln Cys Leu Gly Arg Glu Asp
    850                 855                 860

Leu Gly His Leu Cys Val Trp Ser Asp Gly Cys Arg Leu Arg Gly Pro
865                 870                 875                 880

Leu Gln Pro Met Ala Gly Thr Cys Pro Ala Pro Glu Ile Arg Ala Ile
                885                 890                 895

Glu Pro Leu Ser Gly Pro Leu Asp Gly Gly Thr Leu Leu Thr Ile Arg
            900                 905                 910

Gly Arg Asn Leu Gly Arg Arg Leu Ser Asp Val Ala His Gly Val Trp
        915                 920                 925

Ile Gly Gly Val Ala Cys Glu Pro Leu Pro Asp Arg Tyr Thr Val Ser
    930                 935                 940

Glu Glu Ile Val Cys Val Thr Gly Pro Ala Pro Gly Pro Leu Ser Gly
945                 950                 955                 960

Val Val Thr Val Asn Ala Ser Lys Glu Gly Lys Ser Arg Asp Arg Phe
                965                 970                 975

Ser Tyr Val Leu Pro Leu Val His Ser Leu Glu Pro Thr Met Gly Pro
            980                 985                 990

Lys Ala Gly Gly Thr Arg Ile Thr  Ile His Gly Asn Asp  Leu His Val
        995                 1000                1005

Gly Ser  Glu Leu Gln Val Leu  Val Asn Asp Thr Asp  Pro Cys Thr
    1010                1015                1020
```

```
Glu Leu  Met Arg Thr Asp Thr  Ser Ile Ala Cys Thr  Met Pro Glu
    1025                 1030                 1035

Gly Ala  Leu Pro Ala Pro Val  Pro Val Cys Val Arg  Phe Glu Arg
    1040                 1045                 1050

Arg Gly  Cys Val His Gly Asn  Leu Thr Phe Trp Tyr  Met Gln Asn
    1055                 1060                 1065

Pro Val  Ile Thr Ala Ile Ser  Pro Arg Arg Ser Pro  Val Ser Gly
    1070                 1075                 1080

Gly Arg  Thr Ile Thr Val Ala  Gly Glu Arg Phe His  Met Val Gln
    1085                 1090                 1095

Asn Val  Ser Met Ala Val His  His Ile Gly Arg Glu  Pro Thr Leu
    1100                 1105                 1110

Cys Lys  Val Leu Asn Ser Thr  Leu Ile Thr Cys Pro  Ser Pro Gly
    1115                 1120                 1125

Ala Leu  Ser Asn Ala Ser Ala  Pro Val Asp Phe Phe  Ile Asn Gly
    1130                 1135                 1140

Arg Ala  Tyr Ala Asp Glu Val  Ala Val Ala Glu Glu  Leu Leu Asp
    1145                 1150                 1155

Pro Glu  Glu Ala Gln Arg Gly  Ser Arg Phe Arg Leu  Asp Tyr Leu
    1160                 1165                 1170

Pro Asn  Pro Gln Phe Ser Thr  Ala Lys Arg Glu Lys  Trp Ile Lys
    1175                 1180                 1185

His His  Pro Gly Glu Pro Leu  Thr Leu Val Ile His  Lys Glu Gln
    1190                 1195                 1200

Asp Ser  Leu Gly Leu Gln Ser  His Glu Tyr Arg Val  Lys Ile Gly
    1205                 1210                 1215

Gln Val  Ser Cys Asp Ile Gln  Ile Val Ser Asp Arg  Ile Ile His
    1220                 1225                 1230

Cys Ser  Val Asn Glu Ser Leu  Gly Ala Ala Val Gly  Gln Leu Pro
    1235                 1240                 1245

Ile Thr  Ile Gln Val Gly Asn  Phe Asn Gln Thr Ile  Ala Thr Leu
    1250                 1255                 1260

Gln Leu  Gly Gly Ser Glu Thr  Ala Ile Ile Val Ser  Ile Val Ile
    1265                 1270                 1275
```

```
Cys Ser  Val Leu Leu Leu Leu  Ser Val Val Ala Leu  Phe Val Phe
    1280                 1285                 1290

Cys Thr  Lys Ser Arg Arg Ala  Glu Arg Tyr Trp Gln  Lys Thr Leu
    1295                 1300                 1305

Leu Gln  Met Glu Glu Met Glu  Ser Gln Ile Arg Glu  Glu Ile Arg
    1310                 1315                 1320

Lys Gly  Phe Ala Glu Leu Gln  Thr Asp Met Thr Asp  Leu Thr Lys
    1325                 1330                 1335

Glu Leu  Asn Arg Ser Gln Gly  Ile Pro Phe Leu Glu  Tyr Lys His
    1340                 1345                 1350

Phe Val  Thr Arg Thr Phe Phe  Pro Lys Cys Ser Ser  Leu Tyr Glu
    1355                 1360                 1365

Glu Arg  Tyr Val Leu Pro Ser  Gln Thr Leu Asn Ser  Gln Gly Ser
    1370                 1375                 1380

Ser Gln  Ala Gln Glu Thr His  Pro Leu Leu Gly Glu  Trp Lys Ile
    1385                 1390                 1395

Pro Glu  Ser Cys Arg Pro Asn  Met Glu Glu Gly Ile  Ser Leu Phe
    1400                 1405                 1410

Ser Ser  Leu Leu Asn Asn Lys  His Phe Leu Ile Val  Phe Val His
    1415                 1420                 1425

Ala Leu  Glu Gln Gln Lys Asp  Phe Ala Val Arg Asp  Arg Cys Ser
    1430                 1435                 1440

Leu Ala  Ser Leu Leu Thr Ile  Ala Leu His Gly Lys  Leu Glu Tyr
    1445                 1450                 1455

Tyr Thr  Ser Ile Met Lys Glu  Leu Leu Val Asp Leu  Ile Asp Ala
    1460                 1465                 1470

Ser Ala  Ala Lys Asn Pro Lys  Leu Met Leu Arg Arg  Thr Glu Ser
    1475                 1480                 1485

Val Val  Glu Lys Met Leu Thr  Asn Trp Met Ser Ile  Cys Met Tyr
    1490                 1495                 1500

Ser Cys  Leu Arg Glu Thr Val  Gly Glu Pro Phe Phe  Leu Leu Leu
    1505                 1510                 1515

Cys Ala  Ile Lys Gln Gln Ile  Asn Lys Gly Ser Ile  Asp Ala Ile
    1520                 1525                 1530
```

```
Thr Gly  Lys Ala Arg Tyr Thr  Leu Ser Glu Glu Trp  Leu Leu Arg
    1535                 1540                 1545

Glu Asn  Ile Glu Ala Lys Pro  Arg Asn Leu Asn Val  Ser Phe Gln
    1550                 1555                 1560

Gly Cys  Gly Met Asp Ser Leu  Ser Val Arg Ala Met  Asp Thr Asp
    1565                 1570                 1575

Thr Leu  Thr Gln Val Lys Glu  Lys Ile Leu Glu Ala  Phe Cys Lys
    1580                 1585                 1590

Asn Val  Pro Tyr Ser Gln Trp  Pro Arg Ala Glu Asp  Val Asp Leu
    1595                 1600                 1605

Gly Gly  Ser Pro Pro Ala His  Arg Ala Thr Ser Phe  Gly Thr Trp
    1610                 1615                 1620

Thr Thr  Pro Gln Trp Trp Lys  Thr Ala Ala Arg Ser  Leu Thr Arg
    1625                 1630                 1635

Trp Pro  Ile Thr Arg Ser Leu  Lys Val Pro Pro Trp  Pro
    1640                 1645                 1650
```

<210> 151
<211> 207
<212> PRT
<213> Homo sapiens

<400> 151

```
Met Ala Pro Phe Glu Pro Leu Ala Ser Gly Ile Leu Leu Leu Leu Trp
1               5                  10                  15

Leu Ile Ala Pro Ser Arg Ala Cys Thr Cys Val Pro Pro His Pro Gln
            20                  25                  30

Thr Ala Phe Cys Asn Ser Asp Leu Val Ile Arg Ala Lys Phe Val Gly
        35                  40                  45

Thr Pro Glu Val Asn Gln Thr Thr Leu Tyr Gln Arg Tyr Glu Ile Lys
    50                  55                  60

Met Thr Lys Met Tyr Lys Gly Phe Gln Ala Leu Gly Asp Ala Ala Asp
65                  70                  75                  80

Ile Arg Phe Val Tyr Thr Pro Ala Met Glu Ser Val Cys Gly Tyr Phe
                85                  90                  95

His Arg Ser His Asn Arg Ser Glu Glu Phe Leu Ile Ala Gly Lys Leu
            100                 105                 110
```

```
Gln Asp Gly Leu Leu His Ile Thr Thr Cys Ser Phe Val Ala Pro Trp
        115                 120                 125

Asn Ser Leu Ser Leu Ala Gln Arg Arg Gly Phe Thr Lys Thr Tyr Thr
    130                 135                 140

Val Gly Cys Glu Glu Cys Thr Val Phe Pro Cys Leu Ser Ile Pro Cys
145                 150                 155                 160

Lys Leu Gln Ser Gly Thr His Cys Leu Trp Thr Asp Gln Leu Leu Gln
                165                 170                 175

Gly Ser Glu Lys Gly Phe Gln Ser Arg His Leu Ala Cys Leu Pro Arg
            180                 185                 190

Glu Pro Gly Leu Cys Thr Trp Gln Ser Leu Arg Ser Gln Ile Ala
        195                 200                 205
```

<210> 152
<211> 2101
<212> PRT
<213> Homo sapiens

<400> 152

```
Met Thr Leu His Ala Thr Arg Gly Ala Ala Leu Leu Ser Trp Val Asn
1               5                   10                  15

Ser Leu His Val Ala Asp Pro Val Glu Ala Val Leu Gln Leu Gln Asp
            20                  25                  30

Cys Ser Ile Phe Ile Lys Ile Ile Asp Arg Ile His Gly Thr Glu Glu
        35                  40                  45

Gly Gln Gln Ile Leu Lys Gln Pro Val Ser Glu Arg Leu Asp Phe Val
    50                  55                  60

Cys Ser Phe Leu Gln Lys Asn Arg Lys His Pro Ser Ser Pro Glu Cys
65                  70                  75                  80

Leu Val Ser Ala Gln Lys Val Leu Glu Gly Ser Glu Leu Glu Leu Ala
                85                  90                  95

Lys Met Thr Met Leu Leu Leu Tyr His Ser Thr Met Ser Ser Lys Ser
            100                 105                 110

Pro Arg Asp Trp Glu Gln Phe Glu Tyr Lys Ile Gln Ala Glu Leu Ala
        115                 120                 125

Val Ile Leu Lys Phe Val Leu Asp His Glu Asp Gly Leu Asn Leu Asn
    130                 135                 140
```

```
Glu Asp Leu Glu Asn Phe Leu Gln Lys Ala Pro Val Pro Ser Thr Cys
145                 150                 155                 160

Ser Ser Thr Phe Pro Glu Glu Leu Ser Pro Pro Ser His Gln Ala Lys
                165                 170                 175

Arg Glu Ile Arg Phe Leu Glu Leu Gln Lys Val Ala Ser Ser Ser Ser
            180                 185                 190

Gly Asn Asn Phe Leu Ser Gly Ser Pro Ala Ser Pro Met Gly Asp Ile
        195                 200                 205

Leu Gln Thr Pro Gln Phe Gln Met Arg Arg Leu Lys Lys Gln Leu Ala
    210                 215                 220

Asp Glu Arg Ser Asn Arg Asp Glu Leu Glu Leu Glu Leu Ala Glu Asn
225                 230                 235                 240

Arg Lys Leu Leu Thr Glu Lys Asp Ala Gln Ile Ala Met Met Gln Gln
                245                 250                 255

Arg Ile Asp Arg Leu Ala Leu Leu Asn Glu Lys Gln Ala Ala Ser Pro
            260                 265                 270

Leu Glu Pro Lys Glu Leu Glu Glu Leu Arg Asp Lys Asn Glu Ser Leu
        275                 280                 285

Thr Met Arg Leu His Glu Thr Leu Lys Gln Cys Gln Asp Leu Lys Thr
    290                 295                 300

Glu Lys Ser Gln Met Asp Arg Lys Ile Asn Gln Leu Ser Glu Glu Asn
305                 310                 315                 320

Gly Asp Leu Ser Phe Lys Leu Arg Glu Phe Ala Ser His Leu Gln Gln
                325                 330                 335

Leu Gln Asp Ala Leu Asn Glu Leu Thr Glu Glu His Ser Lys Ala Thr
            340                 345                 350

Gln Glu Trp Leu Glu Lys Gln Ala Gln Leu Glu Lys Glu Leu Ser Ala
        355                 360                 365

Ala Leu Gln Asp Lys Lys Cys Leu Glu Glu Lys Asn Glu Ile Leu Gln
    370                 375                 380

Gly Lys Leu Ser Gln Leu Glu Glu His Leu Ser Gln Leu Gln Asp Asn
385                 390                 395                 400

Pro Pro Gln Glu Lys Gly Glu Val Leu Gly Asp Val Leu Gln Leu Glu
                405                 410                 415
```

```
Thr Leu Lys Gln Glu Ala Ala Thr Leu Ala Ala Asn Asn Thr Gln Leu
            420                 425                 430

Gln Ala Arg Val Glu Met Leu Glu Thr Glu Arg Gly Gln Gln Glu Ala
        435                 440                 445

Lys Leu Leu Ala Glu Arg Gly His Phe Glu Glu Glu Lys Gln Gln Leu
    450                 455                 460

Ser Ser Leu Ile Thr Asp Leu Gln Ser Ser Ile Ser Asn Leu Ser Gln
465                 470                 475                 480

Ala Lys Glu Glu Leu Glu Gln Ala Ser Gln Ala His Gly Ala Arg Leu
                485                 490                 495

Thr Ala Gln Val Ala Ser Leu Thr Ser Glu Leu Thr Thr Leu Asn Ala
            500                 505                 510

Thr Ile Gln Gln Gln Asp Gln Glu Leu Ala Gly Leu Lys Gln Gln Ala
        515                 520                 525

Lys Glu Lys Gln Ala Gln Leu Ala Gln Thr Leu Gln Gln Gln Glu Gln
    530                 535                 540

Ala Ser Gln Gly Leu Arg His Gln Val Glu Gln Leu Ser Ser Ser Leu
545                 550                 555                 560

Lys Gln Lys Glu Gln Gln Leu Lys Glu Val Ala Glu Lys Gln Glu Ala
                565                 570                 575

Thr Arg Gln Asp His Ala Gln Gln Leu Ala Thr Ala Ala Glu Glu Arg
            580                 585                 590

Glu Ala Ser Leu Arg Glu Arg Asp Ala Ala Leu Lys Gln Leu Glu Ala
        595                 600                 605

Leu Glu Lys Glu Lys Ala Ala Lys Leu Glu Ile Leu Gln Gln Gln Leu
    610                 615                 620

Gln Val Ala Asn Glu Ala Arg Asp Ser Ala Gln Thr Ser Val Thr Gln
625                 630                 635                 640

Ala Gln Arg Glu Lys Ala Glu Leu Ser Arg Lys Val Glu Glu Leu Gln
                645                 650                 655

Ala Cys Val Glu Thr Ala Arg Gln Glu Gln His Glu Ala Gln Ala Gln
            660                 665                 670

Val Ala Glu Leu Glu Leu Gln Leu Arg Ser Glu Gln Gln Lys Ala Thr
        675                 680                 685
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```
Glu Lys Glu Arg Val Ala Gln Glu Lys Asp Gln Leu Gln Glu Gln Leu
    690                 695                 700

Gln Ala Leu Lys Glu Ser Leu Lys Val Thr Lys Gly Ser Leu Glu Glu
705                 710                 715                 720

Glu Lys Arg Arg Ala Ala Asp Ala Leu Glu Glu Gln Gln Arg Cys Ile
                725                 730                 735

Ser Glu Leu Lys Ala Glu Thr Arg Ser Leu Val Glu Gln His Lys Arg
            740                 745                 750

Glu Arg Lys Glu Leu Glu Glu Glu Arg Ala Gly Arg Lys Gly Leu Glu
        755                 760                 765

Ala Arg Leu Leu Gln Leu Gly Glu Ala His Gln Ala Glu Thr Glu Val
    770                 775                 780

Leu Arg Arg Glu Leu Ala Glu Ala Met Ala Ala Gln His Thr Ala Glu
785                 790                 795                 800

Ser Glu Cys Glu Gln Leu Val Lys Glu Val Ala Ala Trp Arg Asp Gly
                805                 810                 815

Tyr Glu Asp Ser Gln Gln Glu Glu Ala Gln Tyr Gly Ala Met Phe Gln
            820                 825                 830

Glu Gln Leu Met Thr Leu Lys Glu Glu Cys Glu Lys Ala Arg Gln Glu
        835                 840                 845

Leu Gln Glu Ala Lys Glu Lys Val Ala Gly Ile Glu Ser His Ser Glu
    850                 855                 860

Leu Gln Ile Ser Arg Gln Gln Asn Lys Leu Ala Glu Leu His Ala Asn
865                 870                 875                 880

Leu Ala Arg Ala Leu Gln Gln Val Gln Glu Lys Glu Val Arg Ala Gln
                885                 890                 895

Lys Leu Ala Asp Asp Leu Ser Thr Leu Gln Glu Lys Met Ala Ala Thr
            900                 905                 910

Ser Lys Glu Val Ala Arg Leu Glu Thr Leu Val Arg Lys Ala Gly Glu
        915                 920                 925

Gln Gln Glu Thr Ala Ser Arg Glu Leu Val Lys Glu Pro Ala Arg Ala
    930                 935                 940

Gly Asp Arg Gln Pro Glu Trp Leu Glu Glu Gln Gln Gly Arg Gln Phe
945                 950                 955                 960
```

```
Cys Ser Thr Gln Ala Ala Leu Gln Ala Met Glu Arg Glu Ala Glu Gln
                965                 970                 975

Met Gly Asn Glu Leu Glu Arg Leu Arg Ala Ala Leu Met Glu Ser Gln
            980                 985                 990

Gly Gln Gln Gln Glu Glu Arg Gly  Gln Gln Glu Arg Glu  Val Ala Arg
        995                 1000                1005

Leu Thr  Gln Glu Arg Gly Arg  Ala Gln Ala Asp Leu  Ala Leu Glu
    1010                1015                1020

Lys Ala  Ala Arg Ala Glu Leu  Glu Met Arg Leu Gln  Asn Ala Leu
    1025                1030                1035

Asn Glu  Gln Arg Val Glu Phe  Ala Thr Leu Gln Glu  Ala Leu Ala
    1040                1045                1050

His Ala  Leu Thr Glu Lys Glu  Gly Lys Asp Gln Glu  Leu Ala Lys
    1055                1060                1065

Leu Arg  Gly Leu Glu Ala Ala  Gln Ile Lys Glu Leu  Glu Glu Leu
    1070                1075                1080

Arg Gln  Thr Val Lys Gln Leu  Lys Glu Gln Leu Ala  Lys Lys Glu
    1085                1090                1095

Lys Glu  His Ala Ser Gly Ser  Gly Ala Gln Ser Glu  Ala Ala Gly
    1100                1105                1110

Arg Thr  Glu Pro Thr Gly Pro  Lys Leu Glu Ala Leu  Arg Ala Glu
    1115                1120                1125

Val Ser  Lys Leu Glu Gln Gln  Cys Gln Lys Gln Gln  Glu Gln Ala
    1130                1135                1140

Asp Ser  Leu Glu Arg Ser Leu  Glu Ala Glu Arg Ala  Ser Arg Ala
    1145                1150                1155

Glu Arg  Asp Ser Ala Leu Glu  Thr Leu Gln Gly Gln  Leu Glu Glu
    1160                1165                1170

Lys Ala  Gln Glu Leu Gly His  Ser Gln Ser Ala Leu  Ala Ser Ala
    1175                1180                1185

Gln Arg  Glu Leu Ala Ala Phe  Arg Thr Lys Val Gln  Asp His Ser
    1190                1195                1200

Lys Ala  Glu Asp Glu Trp Lys  Ala Gln Val Ala Arg  Gly Arg Gln
    1205                1210                1215
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```
Glu Ala Glu Arg Lys Asn Ser Leu Ile Ser Ser Leu Glu Glu Glu
    1220                1225                1230

Val Ser Ile Leu Asn Arg Gln Val Leu Glu Lys Glu Gly Glu Ser
    1235                1240                1245

Lys Glu Leu Lys Arg Leu Val Met Ala Glu Ser Glu Lys Ser Gln
    1250                1255                1260

Lys Leu Glu Glu Ser Cys Ala Cys Cys Arg Gln Arg Gln Pro Ala
    1265                1270                1275

Thr Val Pro Glu Leu Gln Asn Ala Ala Leu Leu Cys Gly Arg Arg
    1280                1285                1290

Cys Arg Ala Ser Gly Arg Glu Ala Glu Lys Gln Arg Val Ala Ser
    1295                1300                1305

Glu Asn Leu Arg Gln Glu Leu Thr Ser Gln Ala Glu Arg Ala Glu
    1310                1315                1320

Glu Leu Gly Gln Glu Leu Lys Ala Trp Gln Glu Lys Phe Phe Gln
    1325                1330                1335

Lys Glu Gln Ala Leu Ser Thr Leu Gln Leu Glu His Thr Ser Thr
    1340                1345                1350

Gln Ala Leu Val Ser Glu Leu Leu Pro Ala Lys His Leu Cys Gln
    1355                1360                1365

Gln Leu Gln Ala Glu Gln Ala Ala Ala Glu Lys Arg His Arg Glu
    1370                1375                1380

Glu Leu Glu Gln Ser Lys Gln Ala Ala Gly Gly Leu Arg Ala Glu
    1385                1390                1395

Leu Leu Arg Ala Gln Arg Glu Leu Gly Glu Leu Ile Pro Leu Arg
    1400                1405                1410

Gln Lys Val Ala Glu Gln Glu Arg Thr Ala Gln Gln Leu Arg Ala
    1415                1420                1425

Glu Lys Ala Ser Tyr Ala Glu Gln Leu Ser Met Leu Lys Lys Ala
    1430                1435                1440

His Gly Leu Leu Ala Glu Glu Asn Arg Gly Leu Gly Glu Arg Ala
    1445                1450                1455

Asn Leu Gly Arg Gln Phe Leu Glu Val Glu Leu Asp Gln Ala Arg
    1460                1465                1470
```

```
Glu Lys  Tyr Val Gln Glu Leu  Ala Ala Val Arg Ala  Asp Ala Glu
    1475                 1480                 1485

Thr Arg  Leu Ala Glu Val Gln  Arg Glu Ala Gln Ser  Thr Ala Arg
    1490                 1495                 1500

Glu Leu  Glu Val Met Thr Ala  Lys Tyr Glu Gly Ala  Lys Val Lys
    1505                 1510                 1515

Val Leu  Glu Glu Arg Gln Arg  Phe Gln Glu Glu Arg  Gln Lys Leu
    1520                 1525                 1530

Thr Ala  Gln Val Glu Glu Leu  Ser Lys Lys Leu Ala  Asp Ser Asp
    1535                 1540                 1545

Gln Ala  Ser Lys Val Gln Gln  Gln Lys Leu Lys Ala  Val Gln Ala
    1550                 1555                 1560

Gln Gly  Gly Glu Ser Gln Gln  Glu Ala Gln Arg Phe  Gln Ala Gln
    1565                 1570                 1575

Leu Asn  Glu Leu Gln Ala Gln  Leu Ser Gln Lys Glu  Gln Ala Ala
    1580                 1585                 1590

Glu His  Tyr Lys Leu Gln Met  Glu Lys Ala Lys Thr  His Tyr Asp
    1595                 1600                 1605

Ala Lys  Lys Gln Gln Asn Gln  Glu Leu Gln Glu Gln  Leu Arg Ser
    1610                 1615                 1620

Leu Glu  Gln Leu Gln Lys Glu  Asn Lys Glu Leu Arg  Ala Glu Ala
    1625                 1630                 1635

Glu Arg  Leu Gly His Glu Leu  Gln Gln Ala Gly Leu  Lys Thr Lys
    1640                 1645                 1650

Glu Ala  Glu Gln Thr Cys Arg  His Leu Thr Ala Gln  Val Arg Ser
    1655                 1660                 1665

Leu Glu  Ala Gln Val Ala His  Ala Asp Gln Gln Leu  Arg Asp Leu
    1670                 1675                 1680

Gly Lys  Phe Gln Val Ala Thr  Asp Ala Leu Lys Ser  Arg Glu Pro
    1685                 1690                 1695

Gln Ala  Lys Pro Gln Leu Asp  Leu Ser Ile Asp Ser  Leu Asp Leu
    1700                 1705                 1710

Ser Cys  Glu Glu Gly Thr Pro  Leu Ser Ile Thr Ser  Lys Leu Pro
    1715                 1720                 1725
```

```
Arg Thr  Gln Pro Asp Gly Thr  Ser Val Pro Gly Glu  Pro Ala Ser
    1730                  1735                  1740

Pro Ile  Ser Gln Arg Leu Pro  Pro Lys Val Glu Ser  Leu Glu Ser
    1745                  1750                  1755

Leu Tyr  Phe Thr Pro Ile Pro  Ala Arg Ser Gln Ala  Pro Leu Glu
    1760                  1765                  1770

Ser Ser  Leu Asp Ser Leu Gly  Asp Val Phe Leu Asp  Ser Gly Arg
    1775                  1780                  1785

Lys Thr  Arg Ser Ala Arg Arg  Arg Thr Thr Gln Ile  Ile Asn Ile
    1790                  1795                  1800

Thr Met  Thr Lys Lys Leu Asp  Val Glu Glu Pro Asp  Ser Ala Asn
    1805                  1810                  1815

Ser Ser  Phe Tyr Ser Thr Arg  Ser Ala Pro Ala Ser  Gln Ala Ser
    1820                  1825                  1830

Leu Arg  Ala Thr Ser Ser Thr  Gln Ser Leu Ala Arg  Leu Gly Ser
    1835                  1840                  1845

Pro Asp  Tyr Gly Asn Ser Ala  Leu Leu Ser Leu Pro  Gly Tyr Arg
    1850                  1855                  1860

Pro Thr  Thr Arg Ser Ser Ala  Arg Arg Ser Gln Ala  Gly Val Ser
    1865                  1870                  1875

Ser Gly  Ala Pro Pro Gly Arg  Asn Ser Phe Tyr Met  Gly Thr Cys
    1880                  1885                  1890

Gln Asp  Glu Pro Glu Gln Leu  Asp Asp Trp Asn Arg  Ile Ala Glu
    1895                  1900                  1905

Leu Gln  Gln Arg Asn Arg Val  Cys Pro Pro His Leu  Lys Thr Cys
    1910                  1915                  1920

Tyr Pro  Leu Glu Ser Arg Pro  Ser Leu Ser Leu Gly  Thr Ile Thr
    1925                  1930                  1935

Asp Glu  Glu Met Lys Thr Gly  Asp Pro Gln Glu Thr  Leu Arg Arg
    1940                  1945                  1950

Ala Ser  Met Gln Pro Ile Gln  Ile Ala Glu Gly Thr  Gly Ile Thr
    1955                  1960                  1965

Thr Arg  Gln Gln Arg Lys Arg  Val Ser Leu Glu Pro  His Gln Gly
    1970                  1975                  1980
```

```
Pro Gly  Thr Pro Glu Ser Lys  Lys Ala Thr Ser Cys  Phe Pro Arg
    1985                 1990                 1995

Pro Met  Thr Pro Arg Asp Arg  His Glu Gly Arg Lys  Gln Ser Thr
    2000                 2005                 2010

Thr Glu  Ala Gln Lys Lys Ala  Ala Pro Ala Ser Thr  Lys Gln Ala
    2015                 2020                 2025

Asp Arg  Arg Gln Ser Met Ala  Phe Ser Ile Leu Asn  Thr Pro Lys
    2030                 2035                 2040

Lys Leu  Gly Asn Ser Leu Leu  Arg Arg Gly Ala Ser  Lys Lys Ala
    2045                 2050                 2055

Leu Ser  Lys Ala Ser Pro Asn  Thr Arg Ser Gly Thr  Arg Arg Ser
    2060                 2065                 2070

Pro Arg  Ile Ala Thr Thr Thr  Ala Ser Ala Ala Thr  Ala Ala Ala
    2075                 2080                 2085

Ile Gly  Ala Thr Pro Arg Ala  Lys Gly Lys Ala Lys  His
    2090                 2095                 2100
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<210> 153
<211> 169
<212> PRT
<213> Homo sapiens

<400> 153

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Met Ala Ala Glu Ser Leu Pro Phe Ser Phe Gly Thr Leu Ser Ser Trp
1               5                  10                  15

Glu Leu Glu Ala Trp Tyr Glu Asp Leu Gln Glu Val Leu Ser Ser Asp
            20                  25                  30

Glu Asn Gly Gly Thr Tyr Val Ser Pro Pro Gly Asn Glu Glu Glu Glu
        35                  40                  45

Ser Lys Ile Phe Thr Thr Leu Asp Pro Ala Ser Leu Ala Trp Leu Thr
    50                  55                  60

Glu Glu Glu Pro Glu Pro Ala Glu Val Thr Ser Thr Ser Gln Ser Pro
65                  70                  75                  80

His Ser Pro Asp Ser Ser Gln Ser Ser Leu Ala Gln Glu Glu Glu Glu
                85                  90                  95

Glu Asp Gln Gly Arg Thr Arg Lys Arg Lys Gln Ser Gly His Ser Pro
            100                 105                 110

Ala Arg Ala Gly Lys Gln Arg Met Lys Glu Lys Glu Gln Glu Asn Glu
```

115 120 125

Arg Lys Val Ala Gln Leu Ala Glu Glu Asn Glu Arg Leu Lys Gln Glu
130 135 140

Ile Glu Arg Leu Thr Arg Glu Val Glu Ala Thr Arg Arg Ala Leu Ile
145 150 155 160

Asp Arg Met Val Asn Leu His Gln Ala
165

<210> 154
<211> 344
<212> PRT
<213> Homo sapiens

<400> 154

Met Gln Leu Glu Ile Lys Val Ala Leu Asn Phe Ile Ile Ser Tyr Leu
1 5 10 15

Tyr Asn Lys Leu Pro Arg Arg Arg Ala Asp Leu Phe Gly Glu Glu Leu
20 25 30

Glu Arg Leu Leu Lys Lys Lys Tyr Glu Gly His Trp Tyr Pro Glu Lys
35 40 45

Pro Leu Lys Gly Ser Gly Phe Arg Cys Val His Ile Gly Glu Met Val
50 55 60

Asp Pro Val Val Glu Leu Ala Ala Lys Arg Ser Gly Leu Ala Val Glu
65 70 75 80

Asp Val Arg Ala Asn Val Pro Glu Glu Leu Ser Val Trp Ile Asp Pro
85 90 95

Phe Glu Val Ser Tyr Gln Ile Gly Glu Lys Gly Ala Val Lys Val Leu
100 105 110

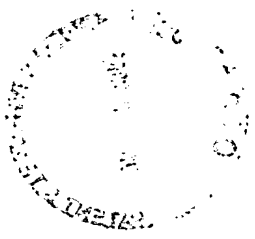
Tyr Leu Asp Asp Ser Glu Gly Cys Gly Ala Pro Glu Leu Asp Lys Glu
115 120 125

Ile Lys Ser Ser Phe Asn Pro Asp Ala Gln Val Phe Val Pro Ile Gly
130 135 140

Ser Gln Asp Ser Ser Leu Ser Asn Ser Pro Ser Pro Ser Phe Gly Gln
145 150 155 160

Ser Pro Ser Pro Thr Phe Ile Pro Arg Ser Ala Gln Pro Ile Thr Phe
165 170 175

Thr Thr Ala Ser Phe Ala Ala Thr Lys Phe Gly Ser Thr Lys Met Lys
180 185 190



```
Lys Gly Gly Gly Ala Ala Ser Gly Gly Gly Val Ala Ser Ser Gly Ala
        195                 200                 205

Gly Gly Gln Gln Pro Pro Gln Gln Pro Arg Met Ala Arg Ser Pro Thr
    210                 215                 220

Asn Ser Leu Leu Lys His Lys Ser Leu Ser Leu Ser Met His Ser Leu
225                 230                 235                 240

Asn Phe Ile Thr Ala Asn Pro Ala Pro Gln Ser Gln Leu Ser Pro Asn
                245                 250                 255

Ala Lys Glu Phe Val Tyr Asn Gly Gly Gly Ser Pro Ser Leu Phe Phe
            260                 265                 270

Asp Ala Ala Asp Gly Gln Gly Ser Gly Thr Pro Gly Pro Phe Gly Gly
        275                 280                 285

Ser Gly Ala Gly Thr Cys Asn Ser Ser Ser Phe Asp Met Ala Gln Val
    290                 295                 300

Phe Gly Gly Gly Ala Asn Ser Leu Phe Leu Glu Lys Thr Pro Phe Val
305                 310                 315                 320

Glu Gly Leu Ser Tyr Asn Leu Asn Thr Met Gln Tyr Pro Ser Gln Gln
                325                 330                 335

Phe Gln Pro Val Val Leu Ala Asn
            340
```